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**GENETICS OF RESISTANCE TO SELENIUM POISONING IN THE
FRUIT FLY, DROSOPHILA MELANOGASTER**

BY

CARL T. HANSEN

A thesis submitted
in partial fulfillment of the requirements for the
degree Master of Science, Department of
Animal Husbandry, South Dakota State
College of Agriculture
and Mechanic Arts

June, 1959

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**GENETICS OF RESISTANCE TO SELENIUM POISONING IN THE
FRUIT FLY, DROSOPHILA MELANOGASTER**

This thesis is approved as a creditable, independent investigation by a candidate for the degree, Master of Science, and acceptable as meeting the thesis requirements for this degree; but without implying that the conclusions reached by the candidate are necessarily the conclusions of the major department.

Thesis Adviser

Head of the Major Department

*

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C. J. H.

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INTRODUCTION

The rare and little known element, selenium, became the object of intensive research when it was discovered as the cause of a mysterious disease which affected livestock in certain areas of the Great Plains. In the course of studying the causes and effects of this disease it was found that soils derived from certain geological formations contained this element which was available to most plants. These plants often contained a sufficient concentration of selenium to be toxic when consumed by livestock. As a result, its presence has caused considerable economic loss to farmers and ranchers in the affected areas since most species of domestic livestock are affected.

Selenium poisoning has been divided into two classes; chronic and sub-acute or acute. The chronic type which is the most prevalent in South Dakota is commonly called "Alkali Disease." Moxon (29) has summarized the general symptoms of the chronic type. It is characterized by (1) dullness and lack of vitality, (2) emaciation and rough hair coat, (3) atrophy of the heart (dish rag heart), (4) atrophy and cirrhosis of the liver, (5) erosion of the long bones, especially in the joints which cause stiffness, (6) loss of the long hair from the mane and tail of horses, from the switch in cattle, and long hair in swine, and (7) soreness and sloughing of the hoofs.

When the cause of "Alkali Disease" was discovered to be selenium, considerable effort was directed towards finding an effective preventive agent. The preventive agents found successful with laboratory animals and swine have not provided the desired protection in beef cattle. They

have either failed in the preliminary tests or they are impractical. The field testing of these preventive agents for range beef cattle has been conducted at the Reed Ranch substation located in a known seleniferous area in south-central South Dakota. When it became apparent that the use of these agents was not completely effective, the selenium studies were directed toward investigating the possibility of breeding for resistance.

To complement the breeding studies at the Reed Ranch station, a series of pilot studies with small animals have been initiated at the Brookings station. The objectives of these studies are to gain experience in working with this problem, to study the effects of selenium on reproduction, and to obtain information on effective breeding plans. This preliminary work can be accomplished in a relatively short time and at low cost while application of the results to the beef cattle studies may save costly errors in either the breeding program or the analysis of the data obtained.

The experiments discussed herein were conducted with the fruit fly, Drosophila melanogaster. This species lends itself very well to a study of this nature since it has a short generation interval, produces a large number of offspring and is best known genetically of the bisexual organisms. The laws of heredity which have been demonstrated to be nearly identical in all bisexual species were to a large extent formulated from studies with D. melanogaster.

The objectives of the experiments presented herein are as follows:

- (1) To study the effects of different concentrations of selenium on reproductive performance. This was necessary in order to determine what concentrations of selenium

should be used in subsequent experiments.

- (2) To determine if natural variation is hereditary and if it can be utilized in breeding for resistance. The rapidity in which insects became resistant to insecticides and the observation of variation in susceptibility of laboratory animals to selenium in controlled experiments made it desirable to determine the feasibility of utilizing this variation in selecting for resistance.
- (3) The relationship of race differences and resistance. There are indications of variation in resistance between different breeds of swine and cattle. The purpose of this experiment was to determine if these differences in susceptibility are the result of morphological differences which identify a particular race or breed of livestock or if this variation is the result of hereditary differences among members of the same breed.
- (4) The relationship of sex to resistance. There are indications that a sex difference may exist in beef cattle since in some herds bulls are affected more severely than cows. The evidence from insecticide studies reveals no sex difference, whereas in mammals sex differences are indicated.
- (5) To obtain estimates of heritability of resistance. The knowledge of heritability is of fundamental importance in utilizing the results of the above experiments in recommending the most effective breeding program.

REVIEW OF LITERATURE

The Effect of Different Levels of Selenium

Early settlers in certain areas of South Dakota reported the presence of a mysterious disease affecting their livestock. It was not definitely established until the 1930's that the element selenium was responsible for what is commonly known as "Alkali Disease." Since then it has been found that levels of selenium between 5 and 15 p.p.m. cause the characteristic symptoms of this disease.

Franke (12) fed samples of corn, wheat and barley from particular localities to 325 white rats. These samples caused the death of 299 of these rats within 100 days. The pathological symptoms exhibited were rapid loss of weight, restricted food intake, hunched posture, roughened fur, staining of the fur around the genitals, paralysis of the hind legs, necrosis of the liver, and lowered hemoglobin levels.

Franke and Potter (13) fed three levels of selenium ad libitum to rats for a period of 359 days with the following results. At 22.3 p.p.m. there were four survivors, at 33.5 p.p.m. one survivor, and at 52.1 p.p.m. all rats died 17 days after the treatment was begun. The symptoms exhibited were similar to those produced by the natural plant toxicant.

Munsell et al. (31) found the threshold dose of selenium in rats to be between 13.0 and 18.4 p.p.m. selenium. The condition of rats receiving 18.4 p.p.m. selenium resembled those receiving a diet containing 9.8 p.p.m. selenium. At the 8.7 p.p.m. concentration, growth was stunted and very few young born. At the 6.6 p.p. m. level, no young

were produced in the second generation. At 3.0 p.p.m. selenium, there was a slight effect on reproduction, however, growth appeared to be normal. No detectable effect on either growth or reproduction was found at the 1.5 p.p.m. level. These workers found wheat containing selenium in a diet for rats had a detrimental effect on growth and reproduction in direct proportion to the amount of selenium provided.

A high correlation was found by Franke et al. (17) between toxicity and selenium content based on the results of feeding 38 seleniferous diets to albino rats. A restriction of food intake was found in every diet containing more than 10 p.p.m. selenium as it occurs in cereals. Concentrations of less than 5 p.p.m. selenium in the diets restricted normal growth.

Poley et al. (34), studying the effects of various levels of selenium in poultry, reported chicks receiving up to 8 p.p.m. selenium in their diets grew as rapidly as those receiving no selenium in their diets. But 10 p.p.m. selenium in the starting rations reduced growth rate and 14 p.p.m. selenium was markedly toxic. No effect of 5 to 14 p.p.m. selenium was found in pullets ranging from 3 to 24 weeks of age.

The effect of selenium on reproduction has considerable economic significance. It has been reported in poultry that selenium reduces hatchability and results in the formation of deformed embryos. In mammals it has been found that the female is apparently more sensitive to the effect of selenium than the male, and evidence has been reported of selenium passage through the fetal membrane to the developing young.

In the mid 1930's investigation of the effect of selenium on the hatchability of fertile hens' eggs was begun by South Dakota workers.

Franke and Tully (14) found 75 percent of the eggs obtained from hens raised in the affected areas which failed to hatch on the twenty-first day contained deformed embryos. Later, Franke and Tully (14) proved that these deformed embryos were the result of selenium by feeding grains containing selenium to hens of a known stock. Franke et al. (15) were able to produce deformed embryos similar to those produced naturally by injecting selenium salts into the air sac of fertile hens' eggs. The greatest number of abnormalities was observed in concentrations of selenium ranging from 0.6 to 0.8 p.p.m. selenium. Above this level development was entirely prevented, and below this level a greater percentage of normal embryos was observed.

The studies of the effect of selenium on reproduction in mammals reported thus far have been concerned with rats. Rosenfeld and Beath (36) adding selenium concentrations of 1.5, 2.5, and 7.5 p.p.m. to the drinking water of a Wistar strain of white rats affected reproduction in proportion to the amount added. The concentrations of 1.5 and 2.5 p.p.m. did not affect reproduction for two generations; however, subsequent reproduction was affected on the 2.5 p.p.m. level. The concentration of 7.5 p.p.m. prevented reproduction in the female but did not affect the fertility of the male. Westfall et al. (43) found evidence of placental transmission of selenium. The fetuses of rats fed organic selenium which contributed 28 percent of the dam's body weight stored about 14 percent of her selenium intake. There was no evidence of deformities as reported in poultry.

Selection and Resistance to Selenium

The results of numerous studies of resistance of insects to chemical poisons and to a lesser degree disease resistance in poultry have indicated considerable variation in the resistance exhibited by an unselected population to the poison or disease in question. These studies have demonstrated that this variation can be utilized in producing progeny which exhibit a greater degree of resistance than the mean of the population from which the resistant parents were selected. Dobzhansky (11) interprets the appearance of DDT resistant strains of houseflies as the result of the population being a mixture of relatively resistant and non-resistant genotypes with the latter having a selective advantage in the absence of DDT. Assuming a parallel situation in other bisexual organisms, it would be possible by selection of resistant genotypes to demonstrate a similar increase, for example, in the resistance to selenium poisoning in beef cattle.

The source of these resistant genotypes may be similar to those observed in the resistance of microorganisms to antibiotics and bacteriophages. Luria and Delbruck (27) found resistant bacteria in a virus susceptible strain which had arisen by mutation independent of the exposure of the bacteria to the virus. Thus, it would be expected that all bacteria would eventually become resistant to the bacteriophage in question. However, Anderson (1) has demonstrated that resistant strains of bacteria require the presence of a certain substance which is not required by the susceptible strain. Consequently, selection keeps the frequency of the resistant bacteria at a very low level when the bacteriophage is absent.

The results of DDT resistance studies with D. melanogaster indicate that progress in the development of resistance is dependent upon the selection pressure to which the population is subjected, but the response to this selection varies from strain to strain. This phenomenon has led several workers to conclude that resistance to DDT is governed by a complex polygenic system rather than by a single or a few genes. Crow (6, 7) found that genes for DDT resistance are initially very rare in the population, since a continuous but accelerating increase in resistance was observed. The author concluded that genes for DDT resistance are not initially advantageous, otherwise the population would have been resistant. King (21, 22) found a differential response between two strains of D. melanogaster to three levels of selection intensity for DDT resistance. One stock exhibited no response, whereas the other stock developed an increase in resistance which was the greatest at the lowest level of selection intensity. Crosses between these two lines gave F_1 and F_3 progeny of intermediate resistance to the parental stocks and F_2 progeny which exhibited less resistance and more variance. It was concluded from these results that the resistant strains had been developed by the consolidation of polygenic factors which are not identical in independently developed strains and in which the constituent factors are not simply additive.

Merrel and Underhill (28) found that the ability of D. melanogaster to become more resistant to DDT was related to the amount of genetic variability in the original population. The development of resistance was directly related to the selection intensity. The higher concentrations of DDT were more effective in increasing resistance and resistance increased

more rapidly after the first few months of selection. Each stock tended to have a somewhat different level of resistance, since some of the controls differed from one another and the resistant stocks also differed from one another as well as from the control stocks. The levels of resistance did not remain static.

Race Differences in Resistance

At the present time there are indications that a relationship may exist between coat color and selenium resistance in beef cattle and swine. Attempts to correlate certain morphological, enzymatic, physiological and behavioral differences with DDT resistance or susceptibility have not been entirely successful. In some cases the correlated responses have been found in traits other than those selected.

Color differences in selenium susceptibility in beef cattle and swine have been reported. Dinkel et al. (10) report the frequency of observations in both experiment station and private herds in which darker colored animals exhibit fewer symptoms of selenium toxicity than lighter colored animals. Wahlstrom et al. (42), studying the effectiveness of arsenalic acid in preventing symptoms of selenium toxicity in Spotted Poland China, Hampshire, and Duroc breeds of swine, found that Durocs exhibited the most severe symptoms, whereas the Spotted Poland China breed did not exhibit any visible symptoms of toxicity.

The instances of morphological, physiological, enzymatic and behavioral differences found between DDT-resistant and susceptible lines of insects are probably of no significance but rather are properties of the different lines tested.

Pratt and Babers (35) correlated cholinesterase activity and oxygen consumption with mortality response in three DDT resistant and susceptible strains of houseflies. Considerable variation in cholinesterase activity was present between the susceptible lines. The difference in oxygen consumption was statistically significant when one susceptible strain was compared with one resistant strain, but the differences were not significant when three susceptible strains were compared with three resistant strains.

A study of behavioral factors made by Sokal and Hunter (40) in which they selected for DDT resistant strains of D. melanogaster on the basis of central or peripheral pupation did not show a perfect correlation. They found the response of the correlated trait lagged one generation behind the selected trait. They suggest modifying genes for the selected trait tended to enhance the correlated trait but not the selected trait.

A detail morphometric analysis of 16 characters in resistant and non-resistant houseflies made by Sokal and Hunter (41) showed that DDT resistance was not correlated with any of the morphological characters studied. They suggest the lack of morphological correlates stem from the fact that the different systems of DDT resistance have evolved between different strains.

There are indications from DDT resistance studies in D. melanogaster that the gene or genes which confer resistance may be in close association with the genes responsible for visible characteristics. Tsukamoto and Ogaki and Ogaki and Tsukamoto cited by Mätcalf (29) found from backcrosses of a Japanese strain that one or several genes for resistance were linked near the Vestigial (vg) gene. In another DDT resistant strain, they found

the gene concerned with resistance located at about 70-80 on the second chromosome map near the Vestigial (67.0) and Scabrous (66.7) genes.

Sex and Resistance

There are indications of a sex difference in the susceptibility of beef cattle to selenium poisoning. Studies of DDT resistance in insects indicate both sexes are equally resistant and contribute an equal amount of resistance to their progeny. Some authors, however, report evidences of maternal effects but concluded that sex linkage is not involved.

Dinkel et al. (10) report one of the ways in which selenium poisoning causes trouble for the rancher is that of crippling the bulls to the extent that they cannot cover their pastures. It appears that the bulls are often more severely crippled than the cows. This may be the result of a sex difference in susceptibility or the result of the cow herd having been carried on selenized pastures for a long enough time to give the operator a chance to select the more resistant females.

The results of reciprocal crosses between DDT resistant and susceptible strains of houseflies made by Harrison (18) show the F_1 progeny were less resistant than the parental stocks, and the performance of the F_2 progeny was still more heterogenous. These results were interpreted as indicating multiple factor inheritance with no evidence for sex linkage.

Norton (32) studying the inheritance of DDT resistance in the housefly found that after eight months of inbreeding all strains showed a marked decline in resistance. Hybridization of the resistant and susceptible strains attenuated the level of resistance. Apart from the actual level of filial tolerance exhibited, there was no difference in the pattern

of resistance transmission, whether emanating from crosses between two resistant strains or between a resistant and susceptible strain. When the progeny of crosses between resistant and susceptible strains were backcrossed to the resistant parent, the level of resistance in the offspring was progressively increased by each backcross. A nearly symmetrical divergent pattern of resistance evolved from similar backcrosses to susceptible parents. There was evidence in the crosses that female parentage had more influence than male parentage, yet this was not considered as evidence for sex linkage.

Crow (7), studying the resistance of hybrids between resistant and susceptible strains of D. melanogaster, found some dominance in genes for resistance. These tests indicated that the majority of the variation is contributed by the autosomes. Sex linked and cytoplasmic factors appeared to play no part.

However, Johnson et al. (20) concluded that DDT resistance in the housefly is apparently under genetic and cytoplasmic control, with the cytoplasmic contribution under genetic control. The resistance of the housefly to DDT is dependent to a large extent on the resistance of its dam because of her relatively large cytoplasmic contribution compared to that of the male. This was not considered as evidence for sex linkage.

Pimental et al. (33), studying the genetic basis of DDT resistance in the housefly by inbreeding and rigid selection, did not obtain a homogenous population. The progeny produced from matings between resistant and susceptible parents were intermediate in their resistance to the parental stocks. These authors also reported that the female parent influenced the resistance of the progeny to a greater extent than the male but concluded resistance was not sex linked.

Busvine and Khan (4) testing for BHC resistance in the housefly by reciprocal matings between two inbred lines, one highly resistant and the other highly susceptible, found that the resistance of the F_1 progeny was intermediate to their parents and the F_2 progeny were more variable.

Lerner (24) discussing the importance of maternal effects in poultry concluded that they may play a significant role. Maternal effects may not be as great in egg laying organisms as in mammals, but they do contribute significantly to various traits since the nutrients supplied by a given dam to a successive series of eggs may show qualitative or quantitative differences from those produced by another dam. Where maternal effects are important, progress made by selection may be reduced in that its net effect is to reduce heritability.

Heritability of Resistance

The criterion used to measure resistance in this study was the number of offspring produced per female parent. Consequently, the estimates obtained herein may reflect egg production rather than selenium resistance. The literature reporting heritability of egg production indicates this trait is highly heritable. The possibility may exist that the mechanism of disease resistance is similar in some respects to the resistance of a chemical poison. The literature on studies of disease resistance in poultry indicates low heritabilities.

Lerner (24), summarizing the available data obtained from studies of the heritability of egg production in poultry reports the majority of the estimates range from 0.30 to 0.35. Shoffner (38) reported an estimate of 0.34 based on data obtained from 751 dam-daughter comparisons using

the method of intra-sire regression. Later, Shoffner and Sloan (39) reported a similar estimate using a method of intra-class correlations. Lerner and Cruden (23), using a method of intra-class correlations between full and half sibs in the University of California flock, found the monthly estimate of heritability ranged from 0.29 to 0.36. These results, taking into consideration species difference, indicate that similar heritabilities may be expected for other egg producing organisms.

The data available on the heritability of disease resistance based on evidence obtained from poultry studies indicate heritabilities of less than 10 percent. Lush et al. (26) studying the records of mortality among more than 20,000 leghorn hens found the following estimates of individual fates by using the directly observed percentages; for total mortality 0.083, for mortality from the leucosis complex 0.053, and 0.034 for other causes than leucosis. A genetic correlation of $\sqrt{0.54}$ was obtained between resistance to the leucosis complex and resistance to death from other causes. The authors interpreted these results to indicate general constitution may play an important part in both kinds of mortality.

Hutt and Cole (19) studying the control of the leucosis complex in poultry found by selection in a genetically resistant line that resistance and ability to lay can be improved concurrently, with the resultant strain having higher viability and greater productivity.

MATERIALS AND METHODS

The experiments discussed herein were conducted in a laboratory established for the purpose of conducting pilot studies with small animals to complement the work with beef cattle at the Reed Ranch station. The methods of culturing the flies were in accordance with procedures which have been found successful in other laboratories. The stock populations were maintained in pint milk bottles. The experimental cultures were maintained in half pint milk bottles capped with either cotton balls or wax caps in which small holes had been punched. The stock cultures were renewed approximately every six weeks or immediately prior to the beginning of a new experiment to ensure vigorous parental stocks. Both the stock and experimental cultures were maintained in an open rack. The temperature was maintained as close to 70 degrees Fahrenheit as possible.

A banana-agar media was used as the basic culture media for the entire study. The selenized media differed only in that liquid sodium selenite was added to the banana-agar media in the desired concentration. The basic formula for the media is as follows:

- 575 cc. water
- 20 grams agar-agar
- 35 grams brewers yeast
- 125 cc. white corn syrup
- 225 cc. crushed ripe banana
- $\frac{1}{2}$ gram mold inhibitor

The first step was to bring the water to a boil at which time the remainder of the ingredients were added along with the selenium. This mixture was allowed to boil for 10 minutes and then poured into sterilized culture bottles to a depth of one-half inch. Strips of paper toweling soaked in

a mold inhibitor solution were then placed into each bottle to allow a dry place for the parents to rest and for pupation of the larvae. The media was allowed to set for at least 24 hours before the cultures were stocked.

The statistical analyses of the results were based upon the mean number of offspring produced per female parent and the ratio of male to female offspring produced in each experimental unit. In the experiments presented in Sections A and B, the mean number of offspring produced per female parent was calculated by dividing the number of female parents removed from each experimental unit at the end of the 10 day laying period into the number of female parents stocked. For the remaining experiments, this figure was obtained by examining the experimental units daily and recording the number of female parents surviving. At the completion of the laying period, the number of female parents surviving were totaled for each experimental unit and divided by 10 to obtain this figure. The ratio of male to female offspring was calculated by dividing the number of female offspring into the number of male offspring produced in each experimental culture.

The factorial design was used since the experiments were conducted for the purpose of studying the effects and interactions of more than one treatment in each experiment. Cochran and Cox (5) summarize some of the instances where a factorial experiment may be suitable as follows:

- (1) In exploratory work where the object is to determine quickly the effects of each of a number of factors over a specified range.
- (2) In investigations of the interactions among the effects of several factors.
- (3) In experiments designed to lead to recommendations that must apply over a wide range of conditions.

The procedural methods used in stocking a new experiment were utilized to minimize all possible variation in parental stocks. The first step in beginning an experiment was to prepare fresh laboratory cultures. Twelve to 24 hours prior to the stocking of the new experiment, the laboratory cultures were emptied to obtain virgin female parents and remove any bias as the result of parental age difference. The parental stocks were examined for physical defects such as broken wings or legs. Three females and two males were placed into each culture bottle which served as the experimental unit. This was done in order to insure against the failure of the experimental unit due to death or sterility of either parent. Immediately after stocking the cultures were placed on their sides for 24 hours to prevent the etherized parents from becoming stuck in the soft media. Parents failing to survive were replaced the next day.

The offspring from the experimental cultures were removed daily in order to obtain an accurate count of the number of offspring produced. The offspring were etherized, examined under a low power microscope, classified according to sex, and counted. The counting period continued until offspring ceased to appear or when it became apparent the second generation offspring were hatching.

There were two sources of bias in the results obtained in these experiments. One source was the media becoming soft and sticky shortly after the parents were stocked. This seemed to be more prevalent in the selenized media. Under these conditions it was difficult for the female to lay her eggs without becoming stuck and drowning. In certain instances this condition contributed to the failure of the experimental unit. Thus, the statistical analyses were conducted using a method outlined by

Snedecor (37) for unequal subclass numbers. The total degrees of freedom for each experiment reflect the actual number of experimental units producing offspring rather than the number making up the experiment. In addition, the soft media made it difficult to obtain accurate counts since the newly emerged offspring became stuck requiring the use of a teasing needle to remove them from the cultures. The other major source of bias resulted from sampling errors since only an extremely small portion of the possible parents were tested in any one experiment. Thus, individuals may have been included who were either more resistant or susceptible than the mean of the population. The number of replications which would have helped in circumventing this problem was limited by the facilities available. However, each experiment was made up of at least four replications.

Twelve of the 15 races maintained in the laboratory were utilized in the various phases of this study. The wild races tested were the Ames-II, Oregon-H, and Canton-S obtained from Iowa State College, Ames, Iowa. A wild race designated as the Turtox in these experiments was obtained from the General Biological Supply House, Chicago, Illinois. The race designated as the Brookings was captured locally just prior to the beginning of these experiments. The mutant races represented three of the four D. melanogaster chromosomes. The first chromosome was represented by the Bar eye (B), Apricot eye (W^a), Blood eye (W^{bl}), and the Yellow body (y); the second chromosome by the Vestigial wing (vg), and the Black body (b); and the third chromosome by the Ebony (e) mutant. Bridges and Brehme (3) present a complete description of these mutants.

EXPERIMENTAL RESULTS

A. The Effect of Different Levels of Selenium

The initial experiment of this study was conducted for the purpose of determining the toxicity of selenium on two races of D. melanogaster. The results of studies with laboratory animals and poultry by Franks et al. (15) and Poley et al. (34) indicate selenium concentrations of 10 p.p.m. are toxic. Munsell et al. (31) observed symptoms of selenium toxicity in rats in almost direct proportion to the amount of selenium provided in their diets. The experiment consisted of determining the toxicity of four concentrations: 0, 5, 10, and 15 p.p.m. selenium on the Turtox (a) and the Black body (b) races. The experiment, consisting of six replications, was designed to test the Race and Treatment main effects and the Race X Treatment interaction.

1. Mean Number of Offspring Per Female Parent

The results of this experiment presented in Table I indicate a threshold effect in toxicity between the 10 and 15 p.p.m. treatments, and the presence of a race difference in resistance. The mean square for the Race differences (Table II) indicates there may be real differences in the performance of the two races despite the fact that this source of variation was not significant at either the one or the five percent levels. The mean difference in the performance of these two races was slightly greater than twice the standard error of Race differences. The Treatment effects were highly significant ($P \leq 0.01$). The 5 and 10 p.p.m. treatments were not toxic, whereas the 15 p.p.m. treatment

TABLE I. THE MEAN NUMBER OF OFFSPRING PRODUCED BY TWO RACES ON FOUR TREATMENTS

Races	Treatments in Parts Per Million Selenium				Means
	0	5	10	15	
(A)	67.0	76.1	63.7	23.2	57.5
(b)	53.8	61.5	54.6	34.7	51.1
Means	60.4	68.8	59.2	28.9	54.3
Standard Error of Race Differences				± 2.5	
Standard Error of Treatment Differences				± 3.6	

TABLE II. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON TWO RACES

Source of Variation	Degrees of Freedom	Mean Squares
Total	47	
Replications	5	248.66
Races	1	487.05
Treatments	3	3,655.97**
Races X Treatments	3	442.98*
Pooled Error	35	153.82

** Highly Significant ($P \leq 0.01$)

* Significant ($P \leq 0.05$)

was highly toxic. These results indicate the presence of a threshold effect between 10 and 15 p.p.m. selenium.

The significant ($P \leq 0.05$) Race X Treatment interaction indicates a differential effect of selenium on the two races. The effects of the 5 and 10 p.p.m. treatments when compared to the 0 treatment were not significant for either race, but the (♂) race consistently produced more offspring on these two treatments than the (b) race. The performance of the two races on the 15 p.p.m. treatment was reversed. As a result the magnitude of the difference in performance for the (♂) race was greater between the 0 and 15 p.p.m. treatment than for the (b) race.

2. The Ratio of Male to Female Offspring

As the concentration of selenium was increased there was a tendency for more male than female offspring to be produced as indicated by the ratios of male to female offspring presented in Table III. The Analysis of Variance of these ratios (Table IV) show the Race differences to be highly significant ($P \leq 0.01$). The (b) race consistently produced a greater proportion of male offspring, whereas the (♂) race produced a greater proportion of female offspring. The Mean Square value for the Race differences while not significant indicates there may be some effect of races on the sex ratio of offspring produced.

TABLE III. THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY TWO RACES ON FOUR TREATMENTS

Races	Treatments in Parts Per Million Selenium				Means
	0	5	10	15	
(f)	0.78	0.87	0.87	0.97	0.88
(b)	1.04	0.98	1.05	1.19	1.06
Means	0.91	0.94	0.96	1.08	0.97
Standard Error of Race Differences				± 0.04	
Standard Error of Treatment Differences				± 0.06	

TABLE IV. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY TWO RACES

Source of Variation	Degrees of Freedom	Mean Squares
Total	47	
Replications	5	149.67
Races	1	4,294.08**
Treatments	3	708.50
Races X Treatments	3	118.02
Pooled Error	35	393.63

** Highly Significant ($P \leq 0.01$)

B. The Effectiveness of Selection For Resistance

Since selection is the most powerful tool the breeder has to alter the frequency of the desired genotypes, a series of experiments were conducted to study the subsequent performance of flies subjected to varying selection intensities. Laboratory studies of insecticide resistance indicate the presence of considerable variation in the resistance of insects to chemical poisons. The results of these studies indicate the chemical acts as a selective agent favoring the resistant genotypes. In addition these studies have further indicated the resistance exhibited by the progeny to this selective agent is related to the selection intensity to which their parents were subjected. Assuming that selenium acts as a selective agent in a manner similar to the chemical poisons, the resistance of the progeny to selenium should be proportional to the intensity of selection to which their parents have been subjected.

These experiments were designed to compare the subsequent performance of parents subjected to varying selection intensities on different concentrations of selenium for several generations. The outline of these experiments are as follows: The first step was to obtain G_1 progeny raised on 0, 5, 10 and 15 p.p.m. selenium treatments. The letter "G" with the appropriate subscript denotes generation number. The parents for these offspring were to be obtained from the random mating laboratory stocks. A random sample of the G_1 progeny from each treatment was then mated to produce the G_2 progeny on four similar treatments. The G_3 and subsequent progeny were to be produced on the same treatment as their parents. The number of G_1 progeny produced would be an indication of the

natural variation present in the resistance of the unselected parents. The number of G_2 progeny produced on each of the treatments will indicate the effectiveness of selection in the previous generation. The performance of the G_3 and subsequent generations will indicate the effectiveness of the original selection after one or more generations. However, due to the lack of suitable facilities, the experiment was concluded at the completion of the third generation.

The experiment in which the G_1 progeny were produced was designed similar to the experiment presented in Section A. The experiments in which the G_2 and subsequent generations were produced consisted of four replications and were designed to test the Parental Source and Treatment main effects and the Parental Source treatment interaction. To gather additional information of the relationship of body color and resistance, the Turtex (\nearrow) and the Black body (b) races were included. Thus, the Race main effects and Race X Parental Source and Race X Treatment interactions were also tested.

1. Mean Number of Offspring Per Female Parent

a. G_1 Generation

The results of this generation presented in Table V indicate a threshold effect similar to that found in the experiment presented in Section A. The variation in the performance of the two races was not significant, as indicated by the Analysis of Variance presented in Table VI. In this experiment however, the (b) race produced more offspring than the (\nearrow) race. The Treatment differences were again highly significant ($P \leq 0.01$). As in Section A, there were no toxic effects of the 5 and 10

TABLE V. THE MEAN NUMBER OF G_1 PROGENY PRODUCED PER FEMALE PARENT BY TWO RACES ON FOUR TREATMENTS

Races	Treatment in Parts Per Million Selenium				Means
	0	5	10	15	
(/)	90.8	91.7	82.0	55.8	80.1
(b)	83.6	85.7	101.0	72.1	86.5
Means	87.2	88.7	91.5	64.0	83.3
Standard Error of Race Differences				± 3.7	
Standard Error of Treatment Differences				± 5.3	

TABLE VI. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON THE G_1 PROGENY PRODUCED BY TWO RACES

Source of Variation	Degrees of Freedom	Mean Squares
Total	47	
Replications	5	450.92
Races	1	365.21
Treatments	3	1,925.04**
Races X Treatments	3	587.94
Pooled Error	35	334.32

** Highly Significant ($P \leq 0.01$)

p.p.m. treatments, whereas the 15 p.p.m. treatment was highly toxic. The performance of the two races on each treatment was similar to that found in Section A. However, the differences were not as great with the result that the Race X Treatment interaction was not significant.

b. G₂ Generation

The effect of the four treatments on the performance of parents from each of the four parental sources indicated selection in the G₁ generation had been effective only at the 15 p.p.m. concentration. The results of this experiment (Table VII) show that the performance of parents from the 15 p.p.m. source was superior to parents from the other sources. The performance of parents from the 0 source was above that of those from either the 5 or 10 p.p.m. sources, with parents from the 10 p.p.m. source producing the least number of offspring per female parent. The Analysis of Variance of these results presented in Table VIII show the Parental Source differences were significant ($P \leq 0.05$). The Treatment effects were not significant. The most offspring were produced on the 5 p.p.m. treatment followed by the 0, 10 and 15 p.p.m. treatments.

The Parental Source X Treatment interaction was not significant. With one exception, the parents from the 15 p.p.m. treatment produced the most offspring per female parent on all treatments. Within the 0 p.p.m. treatment, parents from the selenized sources produced more offspring than parents from the 0 p.p.m. source on all treatments indicating selection for both general fitness and resistance. On the 5 and 10 p.p.m. treatments, parents from the 0 and 15 p.p.m. sources produced more offspring than parents from the 5 and 10 p.p.m. sources. At the 15 p.p.m. treatment, the number of progeny produced increased roughly to the

TABLE VII. THE MEAN NUMBER OF G_2 PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES ON FOUR TREATMENTS

Treat- ments	Races	Parental Sources				Means
		0 p.p.m.	5 p.p.m.	10 p.p.m.	15 p.p.m.	
0 p.p.m.	(f)	46.4	44.4	46.1	58.2	48.8
	(b)	47.1	74.5	53.8	64.8	60.0
	Means	46.7	59.5	49.9	61.5	54.5
5 p.p.m.	(f)	58.2	34.9	49.9	61.5	54.4
	(b)	71.2	57.7	49.0	78.9	53.2
	Means	64.7	46.4	49.5	74.0	57.9
10 p.p.m.	(f)	43.1	31.9	21.0	42.2	34.5
	(b)	77.8	61.2	40.4	60.7	60.0
	Means	60.5	46.5	30.7	51.4	47.3
15 p.p.m.	(f)	26.6	25.9	44.0	40.9	34.3
	(b)	48.8	57.3	52.5	63.4	55.5
	Means	37.7	41.6	48.2	52.2	44.9
Source Means		52.4	48.5	43.9	59.8	51.1
Standard Error of Race Differences					± 2.8	
Standard Error of Treatment Differences					± 3.9	
Standard Error of Parental Source Differences					± 3.9	

intensity of selection by selenium to which the parents had been subjected. The results of this experiment indicate selection for selenium may be feasible.

The Race mean squares were again highly significant ($P \leq 0.01$). The (b) race consistently produced more offspring per female parent on all source-treatment combinations. The Race X Parental Source and the Race X Treatment interactions were not significant.

TABLE VIII. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON G_2 PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES

Source of Variation	Degrees of Freedom	Mean Squares
Total	127	
Replications	3	713.93
Parental Sources	3	1,698.69*
Treatments	3	1,047.16
Parental Sources X Treatments	9	674.31
Races	1	9,261.60**
Races X Parental Sources	3	541.39
Races X Treatments	3	304.25
Parental Sources X Treatments X Races	9	90.56
Pooled Error	93	495.70

** Highly Significant ($P \leq 0.01$)

* Significant ($P \leq 0.05$)

c. G₃ Generation

The results of this experiment presented in Table IX indicate that the progress made by selection in the previous generation was overcome by the treatment effects. The parents originating from the 0, 5 and 10 p.p.m. sources were not affected by 15 p.p.m. selenium to the extent that parents from the 15 p.p.m. sources were. Parents from the 5 and 10 p.p.m. sources produced more offspring than parents from the 0 and 15 p.p.m. sources. These differences as shown by the Analysis of Variance presented in Table X were not significant. The Treatment main effects, however, were highly significant ($P \leq 0.01$). There were significantly more offspring produced on the 5 and 10 p.p.m. treatments than on the 0 and 15 p.p.m. treatments. The 0 and 15 p.p.m. treatments produced essentially the same number of offspring per female parent.

The Parental Source X Treatment interaction was highly significant ($P \leq 0.01$). The performance of stocks from the 0 and 15 p.p.m. sources on the 0 and 15 p.p.m. treatments exhibited little variation, whereas on the 5 and 10 p.p.m. treatments they exhibited considerable variation. Parents from the 10 p.p.m. source produced the most offspring on the 5 p.p.m. treatment and produced fewer offspring on the 10 p.p.m. treatment than parents from the 0 and 5 p.p.m. sources. The parents from the 15 p.p.m. source exhibited the most severe treatment effects. On the 0 p.p.m. treatment they produced more offspring than parents from the other sources. However, as the concentration of selenium was increased their performance was reduced until at the 15 p.p.m. treatment they produced fewer offspring than parents from the 0 p.p.m. source.

TABLE IX. THE MEAN NUMBER OF G_3 PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES ON FOUR TREATMENTS

Treatments	Races	Parental Sources				Means
		0 p.p.m.	5 p.p.m.	10 p.p.m.	15 p.p.m.	
0 p.p.m.	(\nearrow)	32.7	12.4	7.9	29.4	20.6
	(b)	27.8	52.4	46.6	57.9	46.2
Means		30.3	32.4	27.3	43.7	33.4
5 p.p.m.	(\nearrow)	34.6	41.2	43.1	30.9	37.5
	(b)	27.9	36.0	88.9	51.6	51.1
Means		31.3	38.6	66.0	41.3	44.3
10 p.p.m.	(\nearrow)	25.4	43.4	12.1	15.5	24.1
	(b)	55.2	69.3	46.5	52.7	55.9
Means		40.3	56.3	29.3	34.1	40.0
15 p.p.m.	(\nearrow)	17.1	10.5	22.5	16.0	16.6
	(b)	39.4	44.8	33.8	25.4	35.9
Means		28.3	27.7	28.2	20.7	26.2
Source Means		32.5	38.8	37.7	34.9	35.9
Standard Error of Race Differences					\nearrow 2.7	
Standard Error of Treatment Differences					\nearrow 3.5	
Standard Error of Parental Source Differences					\nearrow 3.5	

However, neither the Race X Parental Source nor the Race X Treatment interactions were significant. With the exceptions of parents from the 0 and 5 p.p.m. sources, the (b) race again consistently produced more offspring per female parent than the (\nearrow) race.

TABLE X. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON G₃ PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES

Source of Variation	Degrees of Freedom	Mean Squares
Total	122	
Replications	3	252.20
Parental Sources	3	347.45
Treatments	3	1,782.51**
Parental Sources X Treatments	9	1,171.14**
Races	1	16,340.37**
Races X Parental Sources	3	682.94
Races X Treatments	3	628.07
Parental Sources X Treatments X Races	9	533.81
Pooled Error	88	388.08

** Highly Significant ($P \leq 0.01$)

2. The Ratio of Male to Female Offspring

The ratio of male to female offspring produced per female parent in this three generation study was not affected by either parental source or treatment differences. However, in the G_2 generation the interaction between these two factors did cause significant differences in this ratio. The Race main effects were significant in all three generations.

a. G_1 Generation

The mean ratio of male to female offspring produced per female parent (Table XI) show the (a) race produced a greater proportion of female offspring, whereas the (b) race produced a greater proportion of male offspring. The difference as shown in Table XII was highly significant ($P < 0.01$). Neither the Treatment main effects nor the Races X Treatment interaction were significant. The treatment means do not exhibit a consistent pattern. The ratios for the 0 and 10 p.p.m. treatments indicate a greater proportion of female offspring, whereas for the 5 and 15 p.p.m. treatments a greater proportion of male offspring were produced. The ratios produced by the two races were consistent for all treatments. The (a) race produced a greater proportion of female offspring and the (b) race produced a greater proportion of male offspring.

b. G_2 Generation

The ratios of male to female offspring produced in the G_2 generation reveal no significantly different Parental Source or Treatment effects as shown in Table XIII. The parental source means show that the parents from 0, 5 and 10 p.p.m. sources produced a greater proportion of female offspring, whereas parents from the 15 p.p.m. source produced a greater proportion of male offspring. The mean square for Parental Sources was

TABLE XI. THE RATIO OF MALE TO FEMALE G_1 PROGENY PRODUCED BY TWO RACES ON FOUR TREATMENTS

Races	Treatments in Parts Per Million Selenium				Means
	0	5	10	15	
(f)	0.84	0.94	0.94	0.82	0.88
(b)	1.16	1.04	1.17	1.02	1.10
Means	1.01	0.99	1.05	0.92	0.99
Standard Error of Race Differences				± 0.04	
Standard Error of Treatment Differences				± 0.05	

TABLE XII. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON THE RATIO OF MALE TO FEMALE G_1 PROGENY PRODUCED BY TWO RACES

Source of Variation	Degrees of Freedom	Mean Squares
Total	47	
Replications	5	471.32
Races	1	5,482.69**
Treatments	3	239.96
Races X Treatments	3	325.02
Pooled Error	35	328.18

** Highly Significant ($P \leq 0.01$)

TABLE XIII. THE RATIO OF MALE TO FEMALE G_2 PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES ON FOUR TREATMENTS

Treatments	Races	Parental Sources				
		0 p.p.m.	5 p.p.m.	10 p.p.m.	15 p.p.m.	Means
0 p.p.m.	(f)	0.89	0.91	0.86	0.99	0.93
	(b)	1.04	1.06	0.91	1.27	1.07
	Means	0.97	1.01	0.88	1.13	1.00
5 p.p.m.	(f)	0.80	0.66	0.85	0.87	0.79
	(b)	0.94	1.14	1.14	0.97	1.05
	Means	0.87	0.90	1.00	0.92	0.92
10 p.p.m.	(f)	0.85	0.76	1.00	0.83	0.86
	(b)	1.19	0.94	1.39	0.92	1.11
	Means	1.02	0.85	1.20	0.88	0.98
15 p.p.m.	(f)	0.83	0.78	0.79	1.05	0.86
	(b)	1.07	1.14	0.97	1.34	1.13
	Means	0.95	0.96	0.88	1.20	1.00
Source Means		0.95	0.93	0.99	1.03	0.98
Standard Error of Race Differences					0.03	
Standard Error of Treatment Differences					0.04	
Standard Error of Parental Source Differences					0.04	

not significant as shown in Table XIV. There were no significant differences between the four treatments. The treatment means show an equal number of male and female offspring produced on the 0 and 15 p.p.m. treatments and a slightly greater proportion of female offspring on the 5 and 10 p.p.m. treatments. The mean square for the Parental Source X Treatment interaction was highly significant ($P \leq 0.01$). The means for each parental source-treatment combination show that parents from the 0 and 5 p.p.m. sources produced a greater proportion of female offspring on all treatments.

TABLE XIV. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON THE RATIO OF MALE TO FEMALE G₂ PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES

Source of Variation	Degrees of Freedom	Mean Squares
Total	127	
Replications	3	926.22
Parental Sources	3	635.05
Treatments	3	435.39
Parental Sources X Treatments	9	1,332.79**
Races	1	16,721.23**
Races X Parental Sources	3	126.93
Races X Treatments	3	279.50
Parental Sources X Treatments X Races	9	395.65
Pooled Error	93	484.48

** Highly Significant ($P \leq 0.01$)

The parents from the 10 p.p.m. source produced a greater proportion of female offspring on the 0 and 15 p.p.m. treatments, an equal proportion on the 5 p.p.m. treatment, and a greater proportion of male offspring on the 10 p.p.m. treatment. The parents from the 15 p.p.m. source produced a greater proportion of male offspring on the 0 and 15 p.p.m. treatments, whereas on the 5 and 10 p.p.m. treatments this ratio was reversed.

The differences in this ratio between the two races were again highly significant ($P \leq 0.01$). The Race X Parental Source and the Race X Treatment interactions were not significant. The (f) race, with one exception, produced a greater proportion of female offspring on all combinations, whereas the (b) race again produced a greater proportion of male offspring.

c. G₃ Generation

There were no significant differences resulting from either Parental Source or Treatment main effects. The ratios for the G₃ generation (Table XV) show that parents from all parental sources produced a greater proportion of male offspring. This was particularly true for parents originally from the 15 p.p.m. source. The treatment differences, while not significant, were slightly larger than the Parental Source mean squares as shown in Table XVI. With the exception of the 0 p.p.m. treatment, there was a slightly greater proportion of male offspring produced on the treatments. This ratio showed the greatest difference on the 5 p.p.m. treatment, whereas the variation in this ratio for the remainder of the treatments was rather small. Despite the greater individual differences in this ratio between the parental source-treatment combination than in the previous generation, the Parental Source X Treatment interaction was not significant. The tendency for all parents to produce a greater proportion of male offspring on the 5 p.p.m. treatment is reflected in the treatment means.

The Race differences were again highly significant ($P \leq 0.01$). However, neither the Race X Parental Source nor the Race X Treatment interactions were significant. As in the two previous generations the

TABLE XV. THE RATIO OF MALE TO FEMALE G_3 PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES ON FOUR TREATMENTS

Treat- ments	Races	Parental Sources				
		0 p.p.m.	5 p.p.m.	10 p.p.m.	15 p.p.m.	Means
0 p.p.m.	(f)	0.78	0.94	0.85	0.91	0.87
	(b)	1.38	0.93	1.14	1.22	1.17
	Means	1.08	0.94	1.00	0.95	0.99
5 p.p.m.	(f)	0.97	1.00	0.86	1.20	1.02
	(b)	1.19	1.64	1.57	1.16	1.39
	Means	1.08	1.32	1.10	1.18	1.21
10 p.p.m.	(f)	1.18	0.90	0.99	0.83	0.97
	(b)	1.17	1.10	1.07	0.88	1.08
	Means	1.17	1.00	1.03	0.85	1.02
15 p.p.m.	(f)	0.80	0.91	0.96	1.44	1.03
	(b)	1.02	1.08	1.02	1.14	1.06
	Means	0.91	1.01	0.99	1.29	1.05
Source Means		1.06	1.07	1.06	1.12	1.08
Standard Error of Race Differences					± 0.05	
Standard Error of Treatment Differences					± 0.06	
Standard Error of Parental Source Differences					± 0.06	

(f) race produced a greater proportion of female offspring and the (b) race produced a greater proportion of male offspring, but the differences in this ratio were not as great.

TABLE XVI. THE ANALYSIS OF VARIANCE OF THE EFFECT OF FOUR TREATMENTS ON THE RATIO OF MALE TO FEMALE G₃ PROGENY PRODUCED BY PARENTS FROM FOUR SOURCES

Source of Variation	Degrees of Freedom	Mean Squares
Total	122	
Replications	3	2,325.50
Parental Sources	3	247.59
Treatments	3	2,500.72
Parental Sources X Treatments	9	1,417.07
Races	1	12,996.27**
Races X Parental Sources	3	1,097.91
Races X Treatments	3	1,925.27
Parental Sources X Treatments X Races	9	1,260.55
Pooled Error	92	1,224.61

** Highly Significant ($P \leq 0.01$)

C. Race Differences in Resistance

There is some evidence that variations in selenium resistance may be due to breed differences or to the characteristics which distinguish one breed of livestock from another. Dinkel et al. (10), discussing the selenium research at the Reed Ranch station, suggest the possibility of a relationship existing between selenium resistance and coat color. They discuss the frequencies of reports from both experiment station and private herds of darker colored animals exhibiting fewer and less severe symptoms of selenium toxicity than lighter colored animals. There are also indications of breed differences in swine to selenium susceptibility. Wahlstrom et al. (42) studying the effectiveness of arsanilic acid in preventing symptoms of selenium toxicity in swine found Duroc pigs exhibited more severe symptoms of selenium poisoning than either the Hampshire or the Spotted Poland China breeds. This is of interest since the two breeds characterized by a predominance of black body color exhibited more resistance than the breed characterized by a red body color. However, attempts to correlate insecticide resistance with certain morphological characteristics have not been entirely successful. This suggests that resistance may be the result of either individual differences or that certain breeds may exhibit a greater resistance irrespective of their distinguishing characteristics.

The experiment presented in this section was conducted for the purpose of determining if selenium resistance is the result of individual differences between morphologically similar races of D. melanogaster or if resistance is related to morphological differences expressed by the

mutants used in this study. This experiment consisted of studying the effects of 15 p.p.m. selenium on 12 races for two generations. Five of the 12 races were wild or normal and seven were mutant exhibiting a variety of morphological differences. The wild races were included to determine if variation in selenium resistance exists among races similar in appearance but obtained from different sources. The mutants were included to determine if a relationship existed between morphological difference and resistance. The wild races tested were the Turtox, Ames-II, Oregon-H, Canton-S, and the Brookings. The body color mutants tested were the first chromosome Yellow body (y), the second chromosome Black body (b), and the third chromosome Ebony body (e). A comparison of the performance of these races with the wild races should provide some indication as to the importance of body color in resistance. The other mutants tested were the Blood eye (w^{bl}), Apricot eye (w^a), Bar eye (B), and the Vestigial wing (vg).

The experiment was designed to study the effect of 15 p.p.m. selenium on the reproductive performance for two generations and to obtain a measure of the relative resistance of these 12 races. It consisted of testing 12 races on two treatments, 0 and 15 p.p.m. selenium, for two generations replicated four times in each generation. A comparison of the mean number of offspring produced by these 12 races on the 15 p.p.m. treatment with that of the 0 p.p.m. treatment was used as a measure of reproductive fitness. The differences in number of offspring produced by each race between the 0 and 15 p.p.m. treatments was used to measure resistance. A correlation between these two criteria was calculated to determine if a relationship between these measurements existed, and if so, to what degree.

1. The Mean Number of Offspring Per Female Parent

There was considerable variation in the performance of 12 races resulting from treatment and temperature differences between the first and second generations. The reproductive performance of the 12 races by generation and treatments (Table XVII) are ranked in descending order of their mean reproductive performance for the two generations. The wide variation ranging from 92.4 to 26.9 offspring per female parent for the Brookings (♂) and Canton-S (♂) races is reflected in the highly significant ($P \leq 0.01$) Race mean square presented in Table XVIII. The highly significant ($P \leq 0.01$) mean square for Treatments is primarily due to 15 p.p.m. selenium reducing the reproductive performance by approximately one-half. Since the mean square for the Race X Treatment interaction was highly significant ($P \leq 0.01$), a genetic and environmental interaction is indicated at least as far as reproductive performance is concerned. A comparison of the two generation treatment means in Table XVII shows that races which produced the greatest number of offspring on the 0 p.p.m. treatment did not necessarily produce the most offspring on the 15 p.p.m. treatment. This indicates that reproductive performance of one particular race in one particular environment is not an indication of its performance in the second environment.

The temperature variations between the first and second generations as well as treatment effects resulted in a differential reproductive performance of the 12 races. During the first generation, the laboratory temperature was below that for optimum reproductive performance, whereas in the second generation this condition was corrected. The first and second generation means of 27.0 and 67.3 offspring per female parent are

TABLE XVII. THE EFFECT OF 15 P.P.M. SELENIUM ON THE REPRODUCTIVE PERFORMANCE OF 12 RACES FOR TWO GENERATIONS

Races	First Generation		Second Generation		Two Generation Mean		Race Means
	0	15	0	15	0	15	
Brookings (f)	71.7	58.9	145.8	93.4	108.8	76.2	92.4
(w ^a)	28.2	16.6	177.0	83.4	102.6	50.0	76.6
Ames (f)	54.6	23.7	91.3	82.7	73.0	53.2	63.1
(b)	14.5	33.5	102.5	67.0	58.5	50.2	54.4
Turtlox (f)	28.4	8.7	90.1	56.5	59.2	32.6	49.2
(e)	55.2	16.8	108.4	11.5	81.8	14.2	48.0
(B)	14.2	14.1	68.9	6.5	41.6	10.3	45.9
(vg)	38.5	21.4	61.8	17.9	50.2	19.6	34.9
(y)	13.8	9.2	83.6	15.2	48.7	12.2	30.4
Oregon-H (f)	41.8	21.2	36.4	23.1	39.1	22.2	30.3
(w ^{bl})	14.2	11.9	79.8	14.7	47.0	12.4	30.1
Canton-S (f)	43.4	0.9	60.7	3.3	52.0	2.1	26.9
Means	35.0	21.1	94.1	40.4	63.5	29.6	48.5

Standard Error of Race Differences ± 6.2
 Standard Error of Treatment Differences ± 2.5

TABLE XVIII. THE ANALYSIS OF VARIANCE OF THE EFFECT OF 15 P.P.M.
SELENIUM ON THE REPRODUCTIVE PERFORMANCE
OF 12 RACES FOR TWO GENERATIONS

Source of Variation	Degrees of Freedom	Mean Squares
Total	184	
Replications	3	2,889.40**
Races	11	2,509.27**
Treatments	1	52,340.18**
Races X Treatments	11	5,595.02**
Generations	1	70,265.19**
Generations X Races	11	8,340.15**
Generations X Treatments	1	20,006.27**
Pooled Error	145	622.47

** Highly Significant ($P \leq 0.01$)

reflected in the highly significant ($P \leq 0.01$) Generation mean squares. The differential reproductive performance of the 12 races between the first and second is reflected in the highly significant ($P \leq 0.01$) Race X Generation mean square. All races exhibited some improvement in the second generation, but there was considerable variation in the magnitude of this increase. There was only one race, the Oregon-H (f), whose performance was essentially the same in both generations. The differential treatment effects are reflected in the highly significant ($P \leq 0.01$) Generation X Treatment mean square. The treatment means of 35.0 and 21.1 offspring per female parent for the first generation, and 94.1 and 40.4

offspring per female parent in the second generation show that the relative toxicity of selenium in the second generation was much more severe than in the first generation.

2. Differences Between the 0 and 15 p.p.m. Treatments

The differences in number of offspring per female parent between the 0 and 15 p.p.m. treatments presented in Table XIX show that these races differed to a considerable extent in their resistance. There were no indications, however, that a relationship is present between morphological differences and resistance since there was as much variation between the wild races as the mutants. The Race mean square (Table XX), while not significant, indicates that real differences may be present between the races. The differences ranged from 8.3 for the (b) race to 62.7 for the (e) race with a mean difference of 33.8 offspring per female parent for the 12 races. The two dark body mutants, the (b) and (e), were respectively the most resistant and susceptible of the 12 races tested.

The temperature variation between the two generations also resulted in considerable differences in the resistance exhibited by the 12 races. The first and second generation means of 15.0 and 52.6 offspring per female parent is reflected in the highly significant ($P < 0.01$) Generation mean square. However, the absence of a significant Generation X Race interaction indicates that an increase in the toxicity of selenium in the second generation was nearly proportional for all races over the first generation. This was true for all races except one, the Oregon-H (f) wild, which exhibited slightly more resistance in the first generation.

TABLE XIX. THE DIFFERENCE BETWEEN THE 0 AND 15 P.P.M. TREATMENTS
FOR THE 12 RACES IN EACH GENERATION

Races	Generation		Means
	I	II	
(b)	-18.9	35.5	8.3
Oregon-H (f)	20.5	13.3	16.9
Ames-II (f)	30.9	34.1	19.7
Turtov (f)	17.3	33.6	25.5
(vg)	17.2	43.9	30.5
(B)	0.1	62.4	31.2
Brookings (f)	12.8	52.4	32.6
(wbl)	2.4	65.0	33.7
(y)	4.6	68.3	36.5
Canton-S (f)	43.2	58.2	50.7
(w ^a)	11.6	93.6	52.6
(e)	38.5	96.8	62.7
Means	15.0	52.6	33.8
Standard Error of Race Differences			± 11.9
Standard Error of Generation Differences			± 4.9

TABLE XX. THE ANALYSIS OF VARIANCE OF THE DIFFERENCES BETWEEN
THE 0 AND 15 P.P.M. TREATMENTS

Source of Variation	Degrees of Freedom	Mean Squares
Total	95	
Replications	3	2,271.07
Races	11	2,193.94
Replications X Races	33	1,838.64
Generations	1	34,028.07**
Generations X Races	11	1,793.63
Generations X Replications	3	4,636.12**
Replications X Races X Generations	33	1,139.23

** Highly Significant ($P \leq 0.01$)

One race, the (b), produced more selenized offspring in the first generation than non-selenized offspring, but in the second generation this pattern was reversed.

A comparison of Tables XVII and XIX in which the races are ranked in descending order of their reproductive fitness or resistance indicates that these two characteristics may not be closely related. The races which ranked high when measured by one characteristic did not necessarily have the same ranking when measured by the second characteristic. A low positive correlation of 0.32 was calculated between reproductive fitness and resistance indicating that reproductive fitness cannot be considered

as an accurate measure of resistance. The relative positions of these 12 races based on their reproductive fitness and resistance indicate that these two criteria may be a matter of individual differences rather than due to distinguishing morphological characteristics.

3. The Ratio of Male to Female Offspring

The ratios of male to female offspring presented in Table XXI for this experiment show a slightly greater proportion of female offspring produced. The significant ($P \leq 0.01$) Race mean square (Table XXII) reflects the variation in this ratio resulting from race differences. The ratios ranged from 1.25 for the (y) race to 0.78 for the (e) race with an experimental mean of 0.96. There was a tendency for a slightly greater proportion of male offspring to be produced on the 15 p.p.m. treatment, but the Treatment mean square was not significant. The significant ($P \leq 0.01$) Generation X Race interaction mean square indicates that generation differences affected the ratio of offspring produced by the 12 races. There was as a general rule a greater proportion of male offspring produced in the first generation, whereas in the second generation a greater proportion of female offspring were produced.

TABLE XXI. THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY 12 RACES ON TWO TREATMENTS FOR TWO GENERATIONS

Races	First Generation		Second Generation		Two Generation Mean		Race Means
	0	15	0	15	0	15	
Brookings (f)	0.93	0.85	0.94	1.02	0.93	0.93	0.93
(w ^a)	1.07	0.56	0.88	0.85	0.98	0.71	0.84
Ames-II (f)	1.01	0.85	1.01	1.20	1.01	1.02	1.02
(b)	0.59	0.96	0.98	0.92	0.76	1.08	0.85
Turtov (f)	0.64	0.83	0.96	0.90	0.83	0.88	0.85
(c)	0.81	0.58	0.89	0.86	0.85	0.72	0.78
(B)	0.94	1.15	1.13	0.79	1.03	0.99	1.02
(vg)	0.98	1.43	1.13	0.78	1.04	1.10	1.08
(y)	0.86	1.75	0.98	1.42	0.92	1.59	1.25
Oregon-H (f)	0.74	0.69	0.81	0.91	0.77	0.83	0.80
(w ^{b1})	0.97	1.50	1.06	0.87	1.01	1.14	1.07
Canton-S (f)	1.21	1.00	0.96	0.84	1.08	0.89	1.03
Means	0.90	1.02	0.93	0.96	0.94	0.99	0.96
Standard Error of Race Differences					Σ 0.09		
Standard Error of Treatment Differences					Σ 0.04		

TABLE XXII. THE ANALYSIS OF VARIANCE OF THE RATIO OF MALE TO FEMALE
OFFSPRING PRODUCED BY 12 RACES ON TWO TREATMENTS

Source of Variation	Degrees of Freedom	Mean Squares
Total	177	
Replications	3	1,029.88
Races	11	3,051.08*
Treatments	1	968.70
Races X Treatments	33	2,118.69
Generations	1	75.16
Generations X Races	11	3,092.50*
Generations X Treatments	1	1,974.27
Pooled Error	138	1,558.02

* Significant ($P \leq 0.05$)

D. Sex and Resistance

One of the troubles resulting from the presence of selenium is that bulls often become crippled to the extent that they cannot cover their pastures. It has been observed in some herds that bulls exhibit more severe symptoms of selenium toxicity than do the cows. This may be the result of a sex difference in susceptibility or of purchasing herd sires from non-seleniferous areas. If the sex differences in susceptibility are due to hereditary differences, they may either be sex limited or sex linked. If the trait is sex limited, it will manifest itself only in one sex. On the other hand, if the trait is sex linked, the heterogametic sex will be affected the most severely. The differences in susceptibility observed may also be the result of purchasing herd sires from areas where selection for selenium resistance has not been practiced, whereas the cow herd may have been subjected to some selection.

In order to determine an effective breeding plan for increasing resistance, the mode of transmission from the parent to the offspring must be determined. If both parents contribute an equal amount of resistance to the offspring, the progeny produced from two resistant parents should exhibit a greater degree of resistance than offspring produced from matings in which only one parent is susceptible. If sex linkage were involved the ratio of male to female offspring produced from reciprocal crosses would differ significantly. If selenium were a sex limited trait, progeny surviving would be limited to only one sex or a preponderance of one sex. The results of studies of the mode of transmission of insecticide resistance indicate both parents contribute an equal amount of resistance to their progeny.

The effect of selenium poisoning reducing the calf crop has considerable economic importance. Selenium may affect reproduction either directly or indirectly. The direct effect may be of restricting the physiological processes required for conception, or indirectly as the failure to carry the calf to term due to inadequate fetal nutrition as the result of injury caused by selenium. There are some indications from the studies of insecticide resistance of possible maternal influence since progeny of resistant females tend to be more resistant than from susceptible females.

Two experiments were conducted to study the influence of the sex of the resistant parent on the resistance of their progeny. Experiment I consisted of comparing the performance of four mating systems, R X R, S X S, R X S and S X R, on 0 and 15 p.p.m. selenium. The letters "R" and "S" denote the resistant and susceptible stocks, respectively. This experiment was conducted for the purpose of studying the mode of transmission of resistance to the progeny. Experiment II consisted of comparing the performance of the same four mating systems in which one or both parents were obtained from non-selenized or selenized stocks. Only the 15 p.p.m. treatment was used in this experiment. The purpose of this experiment was to study the effect of prior selenium exposure on the reproductive performance of the resistant or susceptible male or female parent. The Brookings (✓) race was designated as the resistant stock and Canton-S (✓) race was designated as the susceptible stock. The figure "0" designated the non-selenized stocks obtained from the laboratory populations. The figure "15" designates the selenized stocks which had been maintained on 15 p.p.m. selenium for at least three generations.

It was necessary to repeat both Experiments I and II since the performance of the first experiment in each case was affected by the use of parental stocks which were nearly exhausted. These repeated experiments are designated as Experiments A and B.

1. Mean Number of Offspring Per Female Parent

a. Experiment I

The results of this experiment (Table XXIII) indicate treatment differences and mating system differences contributed to considerable variation in the number of offspring produced per female parent. The Analysis of Variance presented in Table XXIV shows the mean square for Treatments to be highly significant ($P \leq 0.01$). As in the previous experiment, 15 p.p.m. selenium was highly toxic to the progeny produced by the four mating systems. The mean square for Mating Systems was significant ($P \leq 0.05$). The two mating systems in which the female was the resistant parent (R X R and S X R) produced essentially the same number of offspring per female parent. The mating systems in which the female was the susceptible parent exhibited the greatest variation. The S X S mating, averaging 75.2 progeny per female parent, produced the least number of offspring and the R X S mating, averaging 112.0 offspring per female parent, produced the most offspring.

The absence of a significant Treatment X Mating System mean square indicates that treatment effects were equal for the four mating systems. As shown in Table XXIII, the performance of the four mating systems on the 0 p.p.m. treatment was consistently superior to that of the 15 p.p.m. treatment, but within the two treatments there was less variation on the

TABLE XXIII. THE MEAN NUMBER OF OFFSPRING PRODUCED PER FEMALE PARENT BY FOUR MATING SYSTEMS ON TWO TREATMENTS

Treat- ments	Experi- ments	Mating Systems				Means
		R X R	S X S	R X S	S X R	
0 p.p.m.	A	128.1	128.0	202.4	161.0	155.5
	B	131.0	93.0	133.1	109.1	116.7
	Means	129.5	110.5	167.8	135.4	135.8
15 p.p.m.	A	13.8	4.4	16.1	42.8	19.3
	B	104.8	75.4	96.3	66.9	85.8
	Means	59.2	39.9	56.2	56.1	52.6
Experiment Means						
	A	70.9	66.2	109.3	101.9	87.1
	B	117.9	84.2	114.7	88.3	101.3
	Means	94.4	75.2	112.0	95.1	94.2
Standard Error of Treatment Differences					5.6	
Standard Error of Mating System Differences					7.9	

15 p.p.m. treatment. The performance of the four mating systems on the two treatments indicates that hybrid vigor may be present since the means of the reciprocal matings were superior to that of the R X R and S X S matings.

A comparison of the experimental means for Experiments A and B reflect the differences in performance contributed by the use of old laboratory stocks with that of using fresh cultures. While the mean

TABLE XXIV. THE ANALYSIS OF VARIANCE OF THE MEAN NUMBER OF OFFSPRING PRODUCED BY FOUR MATING SYSTEMS ON TWO TREATMENTS

Source of Variation	Degrees of Freedom	Mean Squares
Total	63	
Replications	3	100.29
Treatments	1	110,889.00**
Mating Systems	3	3,612.16*
Treatments X Mating Systems	3	1,886.82
Experiments	1	3,226.24
Experiments X Treatments	1	46,693.88**
Experiments X Mating Systems	3	2,584.85
Pooled Error	48	1,002.78

** Highly Significant ($P \leq 0.01$)
 * Significant ($P \leq 0.05$)

square for Experiments was not significant, the Experiments X Treatments mean square was highly significant ($P \leq 0.01$). There were no deleterious effects noted on the 0 p.p.m. treatment caused by differences in the level of vitality of the parental stocks. In fact there were more offspring produced in Experiment A (155.5) than in Experiment B which produced a mean of 116.7 offspring per female parent. However, on the 15 p.p.m. treatment, the level of vitality of the parental stocks had a very profound effect. The progeny produced by parental stocks in Experiment A were less able to resist the toxic effects of 15 p.p.m. selenium than progeny produced in Experiment B despite the superior reproductive fitness

of parents used in Experiment A. There was also some variation in the performance of the four mating systems between the two experiments, but this did not approach that contributed by treatment differences.

b. Experiment II

The results of this experiment presented in Table XXV indicate that the reproductive performance on 15 p.p.m. selenium is at least partially dependent upon the level of resistance and prior selenium history of the female parent. The means for the parental sources and mating systems in this table show that those combinations in which the female was the non-selenized resistant parent produced more progeny than their opposite counterpart. The Analysis of Variance for this experiment (Table XXVI) shows that the mean square for Parental Sources was not significant and that for Mating Systems was significant ($P \leq 0.01$). The two parental source combinations in which the female was the non-selenized parent (0 X 0 and 15 X 0) produced 43.1 and 45.3 offspring per female parent. Those combinations in which the female was the selenized parent (15 X 15 and 0 X 15) produced 29.0 and 36.6 offspring per female parent. These differences while not significant indicate a definite trend for these two series of parental source combinations. A similar pattern was also present for the mating systems. The two mating systems in which the female was the resistant parent (R X R and S X R) produced 44.4 and 48.2 offspring per female parent, whereas the two mating systems in which she was the susceptible parent (S X S and R X S) produced 27.2 and 30.1 offspring per female parent.

TABLE XXV. THE MEAN NUMBER OF OFFSPRING PRODUCED BY 16 PARENTAL SOURCE-MATING SYSTEMS COMBINATIONS IN TWO EXPERIMENTS

Mating Systems	Experiments	Parental Source Combinations				Means
		0 X 0	15 X 15	0 X 15	15 X 0	
R X R	A	24.6	45.4	27.9	47.4	36.2
	B	80.8	34.4	34.4	60.2	52.5
	Means	52.7	39.9	31.2	53.8	44.4
S X S	A	2.9	1.0	2.2	1.4	2.2
	B	38.1	40.6	23.8	60.9	38.0
	Means	19.8	32.6	16.6	41.1	27.2
R X S	A	4.2	7.3	17.6	7.4	7.9
	B	87.0	28.1	30.9	54.4	50.1
	Means	45.6	17.7	26.4	30.9	30.1
S X R	A	6.5	8.1	50.3	20.6	23.5
	B	68.9	52.2	79.2	85.1	72.9
	Means	43.9	27.0	64.8	52.8	48.2
Experiment Means						
Means	A	11.2	18.8	29.4	23.3	20.7
	B	68.7	37.9	42.1	65.8	53.2
		43.1	29.0	36.6	45.3	38.4
Standard Error of Mating System Differences					5.4	
Standard Error of Parental Source Differences					5.4	
Standard Error of Experiment Differences					3.8	

The absence of a significant Parental Source X Mating System interaction indicates that both of these factors may exert considerable influence in determining whether the offspring survives or dies. The data presented in Table XXV show that those combinations in which the female was the non-selenized resistant parent, with one exception, produced more offspring

TABLE XXVI. THE ANALYSIS OF VARIANCE OF THE MEAN NUMBER OF OFFSPRING
PRODUCED BY 16 PARENTAL SOURCE-MATING SYSTEM
COMBINATIONS IN TWO EXPERIMENTS

Source of Variation	Degrees of Freedom	Mean Squares
Total	108	
Replications	3	787.25
Parental Sources	3	1,478.63
Mating Systems	3	2,754.82*
Parental Sources X Mating Systems	9	959.52
Experiments	1	28,904.99**
Experiments X Parental Sources	3	2,987.64*
Experiments X Mating Systems	3	2,985.41*
Parental Sources X Mating Systems X Experiments	9	361.55
Pooled Error	74	939.99

** Highly Significant ($P \leq 0.01$)
* Significant ($P \leq 0.05$)

than those matings in which she was the non-selenized susceptible parent. However, those matings in which she was the selenized resistant parent, with two exceptions, produced more offspring than those matings in which she was the selenized susceptible parent. These data indicate that contributions from the female may play an important role in determining the fate of the progeny.

The effect in differences of vitality between the parental stocks used for Experiments A and B resulted in significant differences in the performance of the parental source combinations and the mating systems. The highly significant ($P \leq 0.01$) Experiment mean square is reflected in the mean of 20.7 offspring per female parent for Experiment A and 53.2 offspring per female parent for Experiment B. Both the mean squares for Experiment X Parental Source and Experiment X Mating System interactions were significant ($P \leq 0.05$). For the parental source combinations, the reciprocals (O X 15 and 15 X O) produced the most offspring in Experiment A, whereas in Experiment B the combinations in which the female parent was the non-selenized parent (O X O and 15 X O) produced the most offspring per female parent. For the mating systems, the R X R and S X S matings produced the most offspring in both experiments, but the performance of the S X S and R X S matings in Experiment A was extremely poor.

2. The Ratio of Male to Female Offspring

The only source of variation which caused significant differences in this ratio in Experiments I and II was that contributed by differences in parental stocks.

a. Experiment I

The means for the treatments and mating systems presented in Table XXVII exhibit little variation. The mean squares for these two sources of variation (Table XXVIII) were not significant. The ratio for the O p.p.m. treatment indicates an equal proportion of male and female offspring and for the 15 p.p.m. treatment a slightly greater proportion of female

TABLE XXVII. THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY
BY FOUR MATING SYSTEMS ON TWO TREATMENTS

Treat- ments	Experi- ments	Mating Systems				Means
		R X R	S X S	R X S	S X R	
0 p.p.m.	A	1.13	0.88	0.91	0.96	0.97
	B	0.98	1.09	1.01	1.03	1.03
	Means	1.06	0.98	0.96	1.00	1.00
15 p.p.m.	A	0.88	0.89	0.82	0.88	0.87
	B	1.02	0.96	1.12	1.06	1.05
	Means	0.96	0.93	0.97	0.97	0.97
Experiment Means						
	A	1.01	0.88	0.87	0.92	0.92
	B	1.01	1.03	1.06	1.05	1.04
	Means	1.01	0.96	0.96	0.98	0.98
Standard Error of Mating System Differences						± 0.03
Standard Error of Treatment Differences						± 0.05

offspring. With the exception of the R X R mating system, the four mating systems produced a slightly greater proportion of female offspring. The mean square for the Treatment X Mating System interaction was not significant.

The mean square for Experiments was highly significant ($P \leq 0.01$) since there was a greater proportion of female offspring produced in Experiment A and a greater proportion of male offspring produced in

TABLE XXVIII. THE ANALYSIS OF VARIANCE OF THE EFFECT OF TWO TREATMENTS ON THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY FOUR MATING SYSTEMS

Source of Variation	Degrees of Freedom	Mean Squares
Total	63	
Replications	3	217.63
Treatments	1	280.56
Mating Systems	3	92.37
Treatments X Mating Systems	3	84.10
Experiments	1	2,209.00**
Experiments X Treatments	1	588.06
Experiments X Mating Systems	3	262.62
Treatments X Mating Systems X Experiments	3	524.56
Pooled Error	45	363.61

** Highly Significant ($P \leq 0.01$)

Experiment B. The ratio for Experiment A may not be a true measure since only a relatively few offspring were produced. The mean squares for Experiment X Treatment and Experiment X Mating Systems interactions were not significant.

b. Experiment II

The differences in parental source or mating systems did not significantly affect the ratio of male to female offspring produced in

this experiment. The means for the parental source combinations (Table XXIX) show, with the exception of the 15 X 0 combination, a greater proportion of female offspring. However, this variation was not large enough to cause the mean square for Parental Source to be significant as shown in Table XXX. The ratios for the mating systems exhibited somewhat more variation than that for the parental source, but the differences were not significant. Three of the mating systems produced a greater proportion of female offspring and exhibited very little variation between them, whereas the R X S mating produced a greater proportion of male offspring.

The Experiment mean square was not significant; however, a slightly greater proportion of female offspring was produced in Experiment A. The mean square for the Experiment X Parental Source interaction was not significant. However, the Experiment X Mating System interaction was highly significant ($P < 0.01$). The mating systems which produced a greater proportion of female offspring, R X R and S X R, and those which produced a greater proportion of male offspring, S X S and R X S, in Experiment A reversed their ratios in Experiment B.

TABLE XXIX. THE RATIO OF MALE TO FEMALE OFFSPRING PRODUCED BY 16
PARENTAL SOURCE-MATING SYSTEM COMBINATIONS

Treat- ments	Experi- ments	Parental Source Combinations				Means
		0 X 0	15 X 15	0 X 15	15 X 0	
R X R	A	0.73	0.82	0.77	0.92	0.81
	B	1.22	1.01	0.79	1.11	1.03
	Means	0.98	0.91	0.78	1.01	0.92
S X S	A	0.61	1.00	1.77	1.00	1.09
	B	0.77	0.86	0.88	1.24	0.90
	Means	0.74	0.89	1.06	1.16	0.94
R X S	A	0.95	1.38	1.44	1.01	1.16
	B	1.02	0.78	0.91	1.06	0.94
	Means	0.98	1.08	1.09	1.03	1.04
S X R	A	0.88	0.87	0.66	0.85	0.80
	B	1.07	0.90	0.99	0.99	0.99
	Means	1.00	0.89	0.82	0.92	0.90
Experiment Means						
Means	A	0.83	1.03	0.94	0.93	0.94
	B	1.02	0.89	0.89	1.08	0.96
	Means	0.94	0.95	0.91	1.01	0.95
Standard Error of Parental Source Differences				± 0.04		
Standard Error of Mating System Differences				± 0.04		

TABLE XXX. THE ANALYSIS OF VARIANCE OF THE RATIO OF MALE TO
FEMALE OFFSPRING PRODUCED BY 16 PARENTAL SOURCE-MATING
SYSTEM COMBINATIONS

Source of Variation	Degrees of Freedom	Mean Squares
Total	105	
Replications	3	59.92
Parental Sources	3	417.90
Mating Systems	3	1,182.85
Parental Sources X Mating Systems	9	779.39
Experiments	1	220.68
Experiments X Parental Sources	3	1,356.50
Experiments X Mating Systems	3	3,628.82**
Parental Sources X Mating Systems X Experiments	9	1,336.07**
Pooled Error	71	643.71

** Highly Significant ($P \leq 0.01$)

E. Heritability of Resistance

Due to the nature of the range beef cattle operation, breeding for resistance may be the most practical solution to this problem. In order to reach this goal in the shortest time with the least expense, the most effective breeding program must be chosen. The primary consideration in the choice of this breeding program is the heritability of the trait in question. Heritability is one of the most fundamental parameters in a population. It provides a measure of the genetic variation on which selection can operate, and this is an important consideration in choosing an optimum breeding program. The estimates presented herein were calculated to determine what may be expected in similar studies with domestic animals.

Since the criterion used to measure the treatment effects were the mean number of offspring per female parent, the results of the previous experiments may reflect reproductive fitness rather than resistance, per se. Similarly, the estimates obtained herein reflect the heritabilities of reproductive fitness rather than resistance. The heritabilities of reproductive fitness were calculated on both the non-selenized and selenized treatments in order to determine the effect of selenium on this trait. However, there was one experiment in which a measure of resistance was possible. This measure was obtained by calculating the difference in number of offspring produced per female parent between the 0 and 15 p.p.m. treatments.

Two concepts of heritability designated as the narrow sense and the broad sense have been defined by Lush (25). Heritability in the narrow

sense has been defined to include that fraction of the phenotypic variance caused by average or additive effects of genes, whereas heritability in the broad sense has been defined as the fraction of phenotypic differences caused by genetic differences of all kinds. The estimates of reproductive fitness were calculated in both the narrow and broad sense in order to determine the importance of non-additive contributions. The estimate of resistance was calculated in the broad sense.

1. Heritability of Reproductive Fitness in the Narrow Sense

The narrow estimates of reproductive fitness were based on a system of partitioning variance for the determination of heritability by the use of full and half sib methods presented by Lerner (24). The reasoning behind this system is based on the following considerations. Under systems approaching random mating, the genetic variance is obtained from three sources, one-fourth each from constant contributions from the sire and dam and one-half due to chance segregation. Thus, the resulting half-sib correlations must be factored by four and the full-sib correlations doubled in order to obtain the respective estimates. The correlations are derived from the variance components, which in turn are obtained by equating the observed mean squares to their estimates. There is the danger, however, of the inclusion of some of the non-additive genetic and environmental variance in the additive portion. For instance, the value obtained from partitioning the variance between full-sibs may lead to an estimate which will include one-fourth of the variance due to dominance and a small amount of epistasis. The correlation between half-sibs contains one-fourth of the additive genetic variance plus a small amount of the epistatic variance.

A special mating system was necessary to obtain the desired results as a consequence of the short life span of the parents. Two separate matings were required, hereafter referred to as Mating I and Mating II. Mating I consisted of mating two virgin Brookings (♂) females to a Brookings (♂) male. Each female was then placed into a separate culture after being with the male for 48 hours. Mating II consisted of mating a random selection of two full sib daughters of each Mating I female to a Oregon-H (♂) male. Following this mating, the females were then placed into separate cultures. The treatment used for these two matings was 15 p.p.m. selenium. Originally 30 sire groups were begun, however, only 13 survived until the completion of the experiment.

The results of these matings (Table XXI) are based on the number of offspring produced by the Mating II females. For example, the values listed under the column headed "Mating I" are the totals for the reproductive performance of the Mating II females. The Sire totals are based on the total performance of the Mating II females. The total for the Sire groups ranged from 188 to 551 with a mean of 365.3 ± 9.1 offspring. The mean square for Sires presented in Table XXII while not significant indicates that real differences may be present. The total number of offspring produced by the two daughters of each Mating I female ranged from 76 to 298 with a mean of 182.6 ± 6.4 offspring. The mean square for Between Females Within Males was not significant. The variation in the performance of each full-sib daughter of the Mating I females is indicated by the source of variation entitled "Full Sibs." This was used as the error term.

TABLE XXXI. THE REPRODUCTIVE PERFORMANCE OF 13 SIREs AS MEASURED BY THEIR DAUGHTERS

Mating II	Mating I	Sires	Mating II	Mating I	Sires
95 40 100 122	135 222	357	38 74 46 93	112 139	251
57 126 56 91	183 147	330	143 91 92 86	234 178	412
20 56 93 19	76 112	188	93 36 156 125	129 281	410
118 58 93 85	176 178	354	137 135 64 107	272 171	443
165 56 91 72	221 163	384	90 82 114 89	172 203	375
107 104 61 30	211 91	302	102 151 149 149	253 298	551
148 61 88 93	209 181	390	<hr/> Means 91.3 182.6 365.3		

Standard Error of Mating I Differences 4.5
Standard Error of Mating II Differences 6.4
Standard Error of Sire Differences 9.1

TABLE XXXII. THE PARTITIONING OF VARIANCE FOR THE DETERMINATION OF HERITABILITY UNDER RANDOM MATING

Source of Variation	Degrees of Freedom	Mean Squares	Mean Square Expectations
Total	51		
Between Sires	12	2,009.39	$E \neq zD \neq yzS$
Between Females Within Sires	13	1,321.02	$E \neq zD$
Between Full Sibs	26	1,075.83	E

x = the number of sires
 y = the number of females per sire
 z = the number of full sib groups per sire

S = the component of variance between non-sibs (172.09)
 D = the component of variance between females within sires (122.05)
 E = environmental component of variance plus half of the genetic variance (1,075.83)

The total variance is $h^2 + e^2$, of which the genetic portion is h^2 . The estimates of reproductive fitness in the narrow sense were obtained by isolating the components E , D , and S .

$$(a) \text{ the paternal half sib correlation estimate} = \frac{4S}{E + D + S} = 0.50$$

$$(b) \text{ the maternal half sib correlation estimate} = \frac{4D}{E + D + S} = 0.38$$

$$(c) \text{ the full sib estimate} = \frac{2(S + D)}{E + D + S} = 0.43$$

The approximate 90 percent fiducial limits for the sire and dam components derived from the mean squares presented in Table XXXII were calculated by a method outlined by Bross (2). The limits for the sire component were 0 and 357.95 and 0 and 310.01 for the dam component. Since the mean squares from which the components were obtained were not significant, the lower limit is taken at zero. These limits may reflect the presence of sampling errors since only a relatively small sample was included in each component.

The estimates of the heritability of reproductive fitness based on maternal and paternal half sib and full sib correlations are presented in Table XXXII. The estimate of 0.38 based on the maternal half-sib correlations may be a more reliable estimate than the estimate of 0.50 based on the paternal half-sib correlation since the fiducial limits were narrower for the dam component. The estimate of 0.43 based on the full-sib correlation may reflect less magnification due to sampling since this correlation was multiplied by two instead of four as for the half-sib correlations. The differences between the maternal half-sib and the full-sib estimates may include some variation due to dominance.

2. The Estimate of Reproductive Fitness in the Broad Sense

The estimates of reproductive fitness and resistance calculated in the broad sense were based on the results of the experiment presented in Section C. Heritability in the broad sense is based on the assumption that the phenotypic variance is equal to the additive effects of heredity and environment with no correlation between these two factors (Lush, 25). The simplest way in which differences in heredity and environment combine

their effect on the phenotype is $P = H + E$, where they are not measured in their own units but in terms of their effect on the phenotype. Then the phenotypic variance (σP^2) is equal to $\sigma H^2 + \sigma E^2 + 2 \text{Cov}_{HE}$. This is the very simplest when heredity and environment are uncorrelated, then the term " 2Cov_{HE} " becomes zero. The phenotypic variance can then be separated into a portion due to individual differences in heredity and that due to differences in the environment to which the individuals were exposed. Thus, $\frac{\sigma H^2}{\sigma H^2 + \sigma E^2}$ becomes heritability in the broad sense, which is a measure of the fraction of (σP^2) due to differences in the individual's heredity.

The broad estimates of the heritability of reproductive fitness were calculated from the results of the experiment conducted in Section C separated by treatments and generation shown in Table XVII. By separating these results in this manner, estimates for the two treatments in the first and second generations and for the two generations combined were possible. While these estimates are not as reliable as those obtained in the narrow sense, they should provide some indication as to their validity.

There was considerable between as well as within generation variation in the number of progeny produced per female by each of the 12 races on the 0 p.p.m. treatment. The performance of the first generation, presented in Table XVII, Section C, ranged from 71.7 for the Brookings (♂) to 13.8 for the (y) race with a mean of 34.3 \pm 8.9 offspring per female parent for the 12 races. In the second generation, their performance ranged from 145.8 for the Brookings (♂) to 36.4 for the Oregon-H (♂) race with a mean of 92.2 offspring per female parent. The Analysis of

Variance of these results (Table XXXIII) show that the mean square for Races was highly significant ($P \leq 0.01$) in the first generation and significant ($P \leq 0.05$) in the second generation. There was, however, considerable variation in the values for the hereditary and environmental components between the two generations. In the first generation, the hereditary component (Races) was essentially the same as the environmental component (Replications X Races), whereas in the second generation the environmental component was nearly four times greater than the hereditary component. These variations are in turn reflected in the differences in the estimates of heritability presented in Table XXXVI. The first generation estimate was 0.49 ± 0.70 and the second generation estimate was 0.20 ± 0.47 . The reduction in the second generation estimate may be attributed in part to an increase of the environmental component.

The performance of the 12 races on the 15 p.p.m. treatment exhibited a somewhat similar pattern as on the 0 p.p.m. treatment. The mean number of offspring produced in the first generation (Table XVII, Section C) ranged from 58.9 for the Brookings (♂) to 0.9 for the Canton-S (♂) race with a mean of 19.4 ± 7.1 for the 12 races. In the second generation, their performance ranged from 93.4 for the Brookings (♂) to 3.3 for the Canton-S (♂) race with a mean of 40.4 ± 10.6 offspring per female parent for the 12 races. The Analysis of Variance of these results (Table XXIV) show that the Race mean squares were highly significant ($P \leq 0.01$) in both generations. The ratios between the environmental and hereditary components in the first generation were similar to that of the 0 p.p.m. treatment, with the result that the estimate of 0.49 ± 0.70 (Table XXXVI) is exactly the same as for the 0 p.p.m. treatment. In the second generation,

TABLE XXXIII. MEAN SQUARES AND COMPONENTS OF VARIANCE FOR FIRST AND SECOND GENERATION REPRODUCTIVE PERFORMANCE ON THE 0 P.P.M. TREATMENT

Source of Variation	Degrees of Freedom	Mean Squares		Mean Square Components	
		I	II	I	II
Total	47				
Replications	3	204.26	7,615.25		
Races	11	1,537.52**	5,908.68*	304.52	780.95
Replications X Races	33	314.92	2,784.87	314.92	2,784.87

** Highly Significant ($P \leq 0.01$)

* Significant ($P \leq 0.05$)

TABLE XXXIV. MEAN SQUARES AND COMPONENTS OF VARIANCE FOR FIRST AND SECOND GENERATION REPRODUCTIVE PERFORMANCE ON THE 15 P.P.M. TREATMENT

Source of Variation	Degrees of Freedom	Mean Squares		Mean Square Components	
		I	II	I	II
Total	47				
Replications	3	177.95	315.56		
Races	11	982.92**	4,692.63**	182.32	1,061.37
Replications X Races	33	199.64	447.14	199.64	447.14

** Highly Significant ($P \leq 0.01$)

however, the hereditary component was nearly three times as great as the environmental component. An estimate of 0.70 ± 0.84 was calculated for the second generation reproductive performance on the 15 p.p.m. treatment. A comparison of these estimates between the two treatments indicate individual differences in heredity may have been more important on the latter treatment.

The greatest variation in the estimates occurred when the results of the two generations for each treatment were combined. The performance on the 0 p.p.m. treatment (Table XVII, Section C) ranged from 108.8 for the Brookings (♂) to 39.1 for the Oregon-H (♂) race with a mean of 63.5 ± 17.4 offspring per female parent for the 12 races. On the 15 p.p.m. treatment, their performance ranged from 76.2 for the Brookings (♂) to 2.1 for the Canton-S (♂) race with a mean of 29.6 ± 5.5 offspring per female parent. The Analysis of Variance of these results presented in Table XXV show that the Race mean squares for the 0 p.p.m. treatment were not significant, whereas for the 15 p.p.m. treatment they were highly significant ($P \leq 0.01$). The Generation mean squares for both treatments were highly significant ($P \leq 0.01$), and the Race X Generation mean square was highly significant ($P \leq 0.01$) for the 15 p.p.m. treatment. The generation differences have been discussed previously in Section C. A comparison of the hereditary and environmental components for the two treatments indicated that performance on the 0 p.p.m. treatment was influenced to a greater extent by environmental differences, whereas on the 15 p.p.m. treatment hereditary differences were more important. This is, in turn, reflected by estimates calculated for the two treatments. The estimate calculated for the 0 p.p.m. treatment was 0.06 ± 0.24 and the estimate for the 15 p.p.m. treatment was 0.67 ± 0.82 .

TABLE XXXV. MEAN SQUARES AND COMPONENTS OF VARIANCE FOR THE FIRST AND SECOND GENERATION COMBINED REPRODUCTIVE PERFORMANCE ON THE 0 AND 15 P.P.M. TREATMENTS

Source of Variation	Degrees of Freedom	Mean Squares		Mean Square Components	
		I	II	I	II
Total	95				
Replications	3	3,316.63	440.48		
Races	11	4,336.02	4,131.61**	153.23	473.97
Generations	1	80,446.26**	9,752.60**		
Replications X Races	33	561.20	450.22		
Replications X Generations	3	4,502.88	52.83		
Races X Generations	11	3,110.17	1,489.95**		
Replications X Races X Generations	33	2,541.92	229.53	2,541.92	229.53

** Highly Significant ($P \leq 0.01$)

TABLE XXXVI. HERITABILITIES OF REPRODUCTIVE FITNESS ON TWO TREATMENTS, 0 AND 15 P.P.M. SELENIUM

Generation	0 p.p.m.	15 p.p.m.
I	0.49 \pm 0.70	0.49 \pm 0.70
II	0.20 \pm 0.47	0.70 \pm 0.84
I and II	0.006 \pm 0.24	0.67 \pm 0.82

Formula Used for Calculating Standard Error of Estimate

$$\sqrt{\frac{6H}{6E^2 + 6E^2}}$$

3. The Estimates of the Heritability of Resistance in the Broad Sense

The estimates of the heritability of resistance in the broad sense indicate that the differences in the resistance of the 12 races in Section C were due in part to heredity. The measure of resistance was the difference in the mean number of offspring produced on the 0 and 15 p.p.m. treatments. Three estimates were calculated, one for each generation, and an estimate for the two generations combined. The mean squares (Table XXXVII) for Races from which the hereditary components were derived were not significant in either generation. The estimates presented in Table XXXVIII indicate that relative to the second generation, differences in heredity were more important in the first generation. The estimate for the first generation was 0.25 ± 0.50 and the second generation estimate was 0.04 ± 0.24 . The estimate of 0.05 ± 0.24 for the two generations combined indicates the presence of considerable environmental variance. The components for the latter estimate were derived from the Analysis of Variance presented in Table XIX, Section C.

The estimates of heritability of reproductive fitness and resistance in the broad sense indicate that these traits are in part influenced by differences in heredity between individuals. The relative importance of heredity for reproductive fitness varied from treatment to treatment. Environmental variation between the first and second generation affected the heritabilities of resistance as well as those for reproductive fitness.

TABLE XXXVII. MEAN SQUARES AND COMPONENTS OF VARIANCE FOR FIRST AND SECOND GENERATION TREATMENT DIFFERENCES

Source of Variation	Degrees of Freedom	Mean Squares		Mean Square Components	
		I	II	I	II
Total	47				
Replications	3	938.07*	6,407.77*		
Races	11	499.43	3,049.49	133.13	119.36
Replications X Races	33	405.53	2,572.04	405.53	2,572.04

* Significant ($P \leq 0.05$)

TABLE XXXVIII. ESTIMATES OF THE HERITABILITY OF SELENIUM RESISTANCE

Generation		
I	II	I and II
0.25 \pm 0.50	0.04 \pm 0.24	0.05 \pm 0.24

DISCUSSION AND CONCLUSIONS

Selenium, a relatively rare element, is present in certain areas in sufficient quantities to be toxic to most species of farm animals. Its presence affects growth and reproduction to the extent that considerable economic loss has been sustained by farmers and ranchers in the affected areas. The efforts directed toward finding a means of eliminating this problem have not been entirely successful in beef cattle. The experiments presented herein were directed toward investigating the application of animal breeding practices to the control of this problem. The results of these experiments indicated that part of the variation in resistance found is hereditary in origin.

The results of experiments conducted in Sections A, B, C, and D indicate the presence of genes for resistance in an unselected population. In Sections A and B, it was found that the 5 and 10 p.p.m. treatments were not toxic, whereas the 15 p.p.m. treatment was highly toxic. The results of the experiments presented in Sections C and D further demonstrate the toxic effects of 15 p.p.m. selenium.

The response of the unselected parents to the four treatments studied in Sections A and B exhibit a similar pattern. In the experiment presented in Table I, Section A, the parents on the 0 p.p.m. treatment produced a mean of 60.44 compared to 68.77, 59.15, and 28.93 offspring per female parent on the 5, 10, and 15 p.p.m. treatments, respectively. The treatment means for a similar experiment presented in Table V, Section B, were 87.20, 88.68, 91.51, and 64.05 offspring per female parent. These results show some stimulation at the 5 and 10 p.p.m.

treatments, and a significant reduction at the 15 p.p.m. treatment. This evidence indicates that 15 p.p.m. selenium is highly toxic to this species, and that a threshold in toxicity is present between the 10 and 15 p.p.m. treatments.

Additional evidence of the toxicity of the 15 p.p.m. treatment is provided from the results of the experiments presented in Tables XVII and XXIII, Sections C and D. The mean performance of parents on the 0 p.p.m. treatment in Section C was 64.5 offspring compared to 30.2 offspring for the 15 p.p.m. treatment. In Section D, the parents on the 0 p.p.m. treatment produced a mean of 135.8 offspring and parents on the 15 p.p.m. treatment produced a mean of 52.6 offspring per female parent. In both experiments, the Treatment mean squares were highly significant ($P \leq 0.01$).

The results of these experiments are in some respects similar to those obtained in selenium studies with rats and poultry. The major difference in this study occurred at the 5 and 10 p.p.m. treatments. Franke et al. (17) and Munsell et al. (31) studying the effects of selenium on rats and Poley et al. (34) in poultry reported a direct relationship between the amount of selenium provided in the diet and the symptoms of selenium toxicity observed. The observation of the effect of 10 p.p.m. selenium or greater and the indication of threshold effect between the 10 and 15 p.p.m. selenium concentrations in D. melanogaster are similar to those reported in poultry and rat studies. Poley et al. (34) and Munsell et al. (31) reported concentrations of 10 and 13 p.p.m. in poultry and rats, respectively, affected growth rate, reproduction, and increased mortality. Concerning the evidence of a threshold, the same authors

report evidence of a similar situation. In poultry, this level was found to be between 5.0 and 8.0 p.p.m. and between 13.0 and 18.4 p.p.m. in rats.

The results of the experiments presented in Sections A, B, C, and D indicate that the population from which the parental sample was drawn contained a mixture of relatively selenium resistant and susceptible individuals. Since the parents were randomly allotted to the various treatments, it can be assumed that they were of equal reproductive potential. Thus, the treatment differences were the result of selenium acting as a selective agent favoring the most resistant genotype. The highly significant treatment differences indicate that the overall level of resistance was not high in the original stock. This in turn would indicate that genes for resistance did not have a selective advantage in the absence of selenium.

Evidence obtained from resistance studies with bacteria and insects may be helpful in interpreting the results from these experiments. Luria and Delbruck (27) and Demerec (8, 9) discuss the finding of resistant colonies of bacteria and the mutational pattern with which resistance is conferred to these colonies. If a similar mechanism exists in a more complex organism, it could be assumed that a particular population is comprised of a mixture of resistant and non-resistant genotypes. The appearance of DDT resistant strains of houseflies has been interpreted by Dobzhansky (11) as the consequence of the population being a mixture of relatively resistant and non-resistant genotypes with the latter having a selective advantage in the absence of DDT. This may be a partial explanation why the proportion of offspring surviving on the 15 p.p.m. treatment was less than those surviving on the 0 p.p.m. treatment.

Crow (6) concluded from his studies of DDT resistance in D. melanogaster that genes for resistance are initially very rare, otherwise the population would have been resistant. A partial explanation as to why the resistant genotypes exist at such a low frequency in the absence of the selective agent in the case of bacteria has been suggested by Anderson (1). He observed that virus resistant bacteria require a certain substance not required by the virus susceptible strains.

Thus far, it has been found that variation in resistance is present between members of the same population. The next aspect to consider is, can this variation be transmitted to succeeding generations. The results of experiments presented in Section B and C indicate this variation can be transmitted to succeeding generations, though not in the manner expected. The results of the experiments presented in Section B indicate selection initially favors genotype conferring resistance, and as the experiments progressed, genotypes for reproductive fitness. The results of the two generation experiment presented in Section C were inconclusive, probably as the result of temperature differences occurring in the laboratory between the two generations.

The data obtained from the three generation experiment presented in Section B in which the subsequent performance of parents subjected to varying intensities of selection were compared was not consistent with the assumption that selection favors only the genotypes conferring resistance. The number of the G_2 progeny produced on the four treatments tended to be proportional to the intensity of the parental selection. There were fewer G_3 progeny produced by parents subjected to the most intense selection on the selenized treatments than were produced by parents subjected to less intense or no selection.

The number of G_2 progeny produced on the 16 Parental Source-Treatment combinations by the G_1 parents indicate selection favored reproductive fitness as well as resistance. The results of this experiment (Table VII, Section B) show that parental source differences contributed more variation than treatment differences. The parents from the 0 p.p.m. source produced a mean of 52.4 offspring compared to 48.5, 43.9, and 59.8 offspring per female parent for the 5, 10, and 15 p.p.m. sources. The mean square for these differences were significant (Table VIII). It is interesting to note that the performance of parents from the 5 and 10 p.p.m. sources were less than for parents from the 0 and 15 p.p.m. sources despite the fact that more offspring were produced on these treatments in the previous generation. The treatment effects differed from the previous generation primarily due to the increased toxicity of the 10 p.p.m. treatment. A mean of 54.4 offspring was produced on the 0 p.p.m. treatment compared to 57.9, 47.3, and 44.9 offspring per female parent on the 5, 10, and 15 p.p.m. treatments. The mean square for these differences approached significance ($P < 0.05$) indicating the possibility of real differences being present.

Since the Parental Source X Treatment interaction was not significant, selection in the previous generation apparently favored genes for resistance as well as those for reproductive fitness. The parents on the 0 p.p.m. treatment produced a mean of 46.7 offspring compared to 61.5 offspring for parents from the 15 p.p.m. source. This indicates selection for reproductive fitness occurred at the 15 p.p.m. treatment in the previous generation. The response of the parents from the four sources on the 15 p.p.m. treatment indicates, however, that selection favored genes

for resistance as well. The parents from the 0 p.p.m. source produced a mean of 37.7 offspring compared to 41.6, 48.2, and 52.2 offspring per female parent for parents from the 5, 10, and 15 p.p.m. sources. This increase was roughly in proportion to the intensity of parental selection.

There was considerable variation in the performance of parents from the 5 and 10 p.p.m. sources as well as in the effects of the 5 and 10 p.p.m. treatments on progeny produced by parents from the four sources. Since the 5 and 10 p.p.m. treatments stimulated reproduction in the previous generation, genotypes which contributed less resistance or poorer reproductive fitness may have been maintained in the population. The proportion of these less favorable genotypes present may be dependent upon such factors as the state of the culture media and laboratory temperatures at the time the experiment was conducted.

The number of G_3 progeny produced for each of the 16 Parental Source-Treatment combinations presented in Table IX, Section B, indicate the effects of selenium were more severe than in the previous generation. The variation in the number of progeny produced by the parents from the four sources was not significant, whereas the treatment effects were highly significant (Table X). The parental source means of 32.5, 38.8, 37.7, and 35.0 offspring per female parent from the 0, 5, 10, and 15 p.p.m. sources indicate no advantage regardless of prior selection history for reproductive fitness. The treatment effects were similar to those observed in the results of experiments presented in Tables I and V, Sections A and B. The means of 33.4, 44.3, 40.0, and 26.2 offspring per female parent for the 0, 5, 10, and 15 p.p.m. treatments reflect the significant increase of the 5 and 10 p.p.m. treatments over the 0 and 15

p.p.m. treatments. The difference between the 0 and 15 p.p.m. treatments were also significant. Thus, the treatment effects were similar to those observed in unselected populations.

The significant Parental Source X Treatment interaction indicates progeny produced from parents originally subjected to the most severe selection pressure were less resistant than progeny produced from parents subjected to less intense selection. The treatment differences, particularly for the 0 and 15 p.p.m. treatments, were very small for parents from the 0, 5, and 10 p.p.m. sources, whereas for the parents from the 15 p.p.m. source these differences were significant. The significant differences are demonstrated by comparing the mean number of offspring produced by parents from these two sources on the 0, 5, 10, and 15 p.p.m. treatments. The means are as follows: 30.7 and 43.7, 31.3 and 41.3, 40.3 and 34.1, and 28.3 and 20.7. The absence of significant treatment differences for parents from the 0 p.p.m. source may be in part due to the presence of a stress factor which dampened their performance on the 0 p.p.m. treatment but did not affect their performance on the 15 p.p.m. treatment. The significant differences in performance of parents from the 15 p.p.m. source between the 0 and 15 p.p.m. treatments indicate selection in the previous generation may have favored reproductive fitness rather than resistance.

The results of the two generation experiment presented in Section C are inconclusive as to whether selection favors resistance or reproduction in a selenized environment. These may have been influenced by temperature differences existing between the two generations. The results of this experiment (Table XVII and XIX, Section C) present both the mean

number of offspring produced per female parent representing reproductive fitness and differences in the mean number offspring produced between the 0 and 15 p.p.m. treatments representing resistance. There was considerable variation in these two criteria between the first and second generations. Considering reproductive performance, the parents on the 0 p.p.m. treatment produced a mean of 35.0 offspring compared to 21.2 offspring per female on the 15 p.p.m. treatment. However, in the second generation, a mean of 94.1 offspring was produced compared to 40.4 offspring per female parent on the 15 p.p.m. treatment. Since the Generation X Treatment mean square was highly significant ($P \leq 0.01$), a differential effect of selenium was present between the two generations. The differences in resistance exhibited a similar pattern. There was a mean difference of 15.0 offspring for the first generation compared to 52.6 for the second generation.

The results of these two experiments presented in Sections B and C are difficult to interpret as the result of wide generation differences. The evidence did not indicate whether selection in a selenized environment favored resistance and/or reproductive fitness. There were some indications however, that the differences found may be transmitted from generation to generation. The conditions under which these experiments were conducted may have contributed to the variations observed. One source of bias may have been the result of sampling errors, since only an extremely small portion of the total offspring produced was selected to be parents for the next generation. The effects of temperature differences have been discussed previously.

The results obtained herein are similar in some respects to those reported in studies of DDT resistance with D. melanogaster. Evidence

that resistance of the progeny is related to the selection intensity of the parents has been reported in studies of DDT resistance. Merrel and Underhill (28) found a relationship between intensity of parental selection for DDT resistance in D. melanogaster and the resistance exhibited by the offspring. The number of G_2 progeny (Table VII) produced by parents undergoing varying intensities of selection indicate a similar relationship, particularly at the 15 p.p.m. treatment level. However, the performance of the G_3 progeny (Table IX) indicates a general reduction in fertility. The mean of 36.0 offspring per female parent in the G_3 generation compared to 51.1 offspring for the G_2 reflect the fewer number of offspring produced in the parental source-treatment combinations in the G_3 generation. A similar observation, though not in the exact situation, has been reported by King (22) in which he found a general reduction in performance of the F_2 compared to the F_1 and F_3 generations in crosses between DDT resistant and susceptible strains of D. melanogaster.

There were indications from the results of experiments presented in Sections A, B, and C that non-linear interactions between genotype and environment may be important. The results from these experiments indicate that breeding for selenium resistance may be a matter of testing a variety of genotypes in a selenized environment and selecting the superior genotype in that environment instead of aiming toward a population which is characterized by a superior average genotype to all possible genotypes under all environments. Since each of the experiments were designed to test the effect of selenium on one or more races, a test of the Race X Treatment interaction was possible. This interaction is by definition a measure of the failure of the effects of selenium on the races tested

to be linear, assuming each race to be characterized by a unique genotype.

The mean square values for the Race X Treatment interactions tested in the experiments (Tables I and V, Sections A and B) indicate non-linear interactions between genotype and environment between the (f) and (b) races. However, for the G_2 and G_3 generations in Section B, these differences were not as pronounced. The mean for the experiment presented in Section A reflects the significant Race X Treatment mean square (Table II). The (f) and (b) races produced a mean of 67.0 and 53.8 offspring on the 0 p.p.m. treatment and 28.2 and 34.7 offspring per female parent on the 15 p.p.m. treatment. For the experiment presented in Table V, Section B, the (f) and (b) races produced a mean of 90.8 and 83.6 offspring on the 0 p.p.m. treatment and 55.7 and 72.1 offspring per female parent on the 15 p.p.m. treatment. The mean square value (Table VI) for the Race X Treatment interaction indicated real differences may be present. In both the G_2 and G_3 generations, Tables VII and IX, the Race mean squares were highly significant, whereas only in the G_3 generation did the Race X Treatment mean square indicate the presence of genetic-environmental interaction. The results from these experiments indicate the (b) race may be characterized by the presence of genes for superior reproductive fitness in an unfavorable environment.

The evidence of an interaction between genotype and environment in the experiment presented in Section C may be in part due to environmental differences existing between the two experiments. The temperature differences in the laboratory have been discussed elsewhere. The effects of the low temperature in the first generation apparently affected performance to a greater extent on the 0 p.p.m. treatment than on the 15 p.p.m.

treatment. There was a mean of 35.0 offspring compared to 21.1 offspring per female parent produced on the 15 p.p.m. treatment in the first generation. In the second generation, a mean of 94.1 offspring was produced on the 0 p.p.m. treatment compared to 40.4 offspring per female parent on the 15 p.p.m. treatment. These means indicate that selection imposed by selenium may favor reproductive fitness as well as resistance. The difference between the two treatments in the first generation may represent the effect of genes for resistance. However, all races did not respond in a similar manner to the environmental changes with the result that the Race X Generation interaction was highly significant (Table XVIII). Some examples of race differences between the first and second generation (Table XVII) are 24.0 and 84.8 for the (b) race, 31.5 and 29.1 for the Oregon-H (f), 30.0 and 39.8 for the (vg), and for the (W^a) 22.4 and 130.8 offspring per female parent.

This in turn resulted in a significant Race X Treatment interaction. The means for the number of offspring produced on the two treatments by each of the 12 races is presented in Table XVII, Section C. This significant interaction stemmed from the fact that some of the races were above the mean on both treatments, some below the mean, and others approached the mean on the 0 p.p.m., but were well below the mean on the 15 p.p.m. treatment. Some examples of the races whose performance was above the mean on both treatments are the Brookings (f) producing a mean of 108.7 on the 0 p.p.m. treatment and 76.2 offspring per female parent on the 15 p.p.m. treatment, the (W^a) producing 102.6 and 83.4, and the Ames-II (f) producing 72.9 and 53.2 offspring per female parent. The race producing the least number of offspring on both treatments but showing the least

between treatment variation was the Oregon-H (f) producing 39.1 and 22.2 offspring per female parent. An example of a race whose performance was above the mean on the 0 p.p.m. treatment and below the mean on the 15 p.p.m. treatment was the (e) producing 81.8 and 14.1 offspring per female parent. The performance of the Canton-S (f) race, producing 52.1 offspring on the 0 p.p.m. treatment and 2.1 offspring per female parent on the 15 p.p.m. treatment, is an example of a race whose performance on the 0 p.p.m. was near the mean and well below the mean of the 15 p.p.m. treatment. The means were 63.2 for the 0 p.p.m. treatment and 29.5 offspring per female parent for the 15 p.p.m. treatment.

Since there was so much variation in the reproductive performance between the 12 races, a comparison of differences in the mean number of offspring produced on the 0 p.p.m. and 15 p.p.m. treatments is presented in Table XIX. The races are ranked according to the magnitude of these differences. A comparison of this table and Table XVII in which the same races are ranked according to their reproductive performance shows that these two criteria are not necessarily related. Some examples of comparisons between resistance and reproductive performance are as follows: The (b) race which ranked first in resistance ranked fourth in reproductive performance, the Oregon-H (f) race ranked second in resistance and ranked tenth in reproduction, the Canton-S (f) race ranked tenth in resistance and ranked twelfth in reproduction, and the (e) race ranked twelfth in resistance and ranked sixth in reproduction. The relative positions of the 12 races for the two criteria do not indicate genes for resistance and reproduction are the same.

The mean squares for Races presented in Table XX while not significant indicate real differences may be present between the 12 races in their resistance. The differences ranged from 8.2 offspring for the (b) race to 62.7 offspring per female parent for the (e) race. The mean difference for the 12 races was 33.8 offspring per female parent. The positions of the 12 races in Table XIX do not indicate any relationship between visible morphological differences and resistance.

The five (f) races tested exhibited as much variation in both resistance and reproductive fitness as the mutants. Their ranks in resistance (Table XIX) ranged from second to tenth. The Oregon-H (f) race ranked second, the Ames-II third, the Turtox (f) fourth, and the Canton-S tenth. This variation in resistance indicates that within morphologically similar races, a wide diversity of genotypes is present. Thus, breeding for resistance may be a matter of selecting those individuals which possess the genotype conferring the most resistance rather than favoring a particular breed. The variation in resistance of the (f) races may have been the result of each race developing its own unique polygenic system. Observations of a similar nature have been made by King (22) and Merrel and Underhill (28) in their studies of DDT resistance in D. melanogaster.

There were no indications of a relationship existing between body color and selenium resistance found in this study. The three body color mutants, (b), (e), and (y), exhibited considerable variation in their resistance. The two dark body color mutants, (b) and (e), were respectively the most resistant and susceptible of the 12 races tested, and the (y) mutant was intermediate to these two mutants. The two dark body

color mutants would be expected to demonstrate the most resistance if these color genes acted in a manner similar to the dark color genes in swine and beef cattle. Wahlstrom et al. (42) and Dinkel et al. (10) report observations of fewer symptoms of selenium toxicity in the breeds of swine and cattle characterized by a black body color. The results of this experiment indicated that body color and resistance were not related.

There was no evidence of a relationship existing between visible morphological differences characterizing the mutants tested and resistance to selenium. The two eye color mutants, (w^{bl}) and (w^a) ranked eighth and eleventh in their resistance. The (B) mutant ranked sixth and the (vg) mutant ranked fifth. These results are similar to those found by Pratt and Babers (35) and Sokal and Hunter (40, 41) who attempted to correlate physiological and behavioral differences with DDT resistance in houseflies and fruit flies. These workers concluded that different systems of DDT resistance have evolved in separate lines and that modifying genes for the selected trait tended to enhance the correlated trait rather than the selected trait.

Japanese workers have found that genes for DDT resistance in D. melanogaster may be in close association with second chromosome mutants. Tsukamoto and Ogaki and Ogaki and Tsukamoto cited by Metcalf (29) obtained evidence that one or several genes for DDT resistance may be linked with the (vg) gene. Additional studies by the same workers revealed the genes for DDT resistance may be located near the Vestigial (67.0) and Scabrous (66.0) genes on the second chromosome. The second chromosome mutants in this study, (b) and (vg), ranked respectively first and fifth of the 12 races tested. Also, these were the most resistant of the seven mutants tested.

Thus far it has been found in the experiments discussed that selenium exerts a deleterious effect on reproduction. The reports of selenium studies with rats and poultry indicate a similar effect. This aspect of the problem of selenium poisoning has caused considerable economic loss to the farmer and rancher in the affected areas. Since the rancher must purchase his herd sires from non-seleniferous areas, the relationship of sex and resistance assumes considerable importance. Studies of the inheritance of insecticide resistance indicate that sex differences in resistance are not present.

There have been reports of possible sex differences in susceptibility of beef cattle since it has been reported that bulls often exhibit more severe symptoms of selenium toxicity than the cows. The results obtained herein of the effects of selenium provide no evidence of sex differences being present. This was studied by comparing the ratio of male to female offspring produced by parents subjected to these treatments. The ratios appeared to be the result of individual difference rather than treatment differences. There was, however, some evidence of race differences in this ratio in several of the experiments.

There were no significant differences resulting from the effects of the different concentrations of selenium on the ratios of male to female offspring produced in the experiments presented in Sections A, B, C, and D. The ratios of male to female offspring presented in Table III, Section A, indicated a tendency for a greater proportion of male offspring to be produced as the concentrations of selenium increased. The ratios produced on the 0, 5, 10, and 15 p.p.m. treatments were 0.91, 0.94, 0.96, and 1.08. However, the Treatment mean squares (Table IV) were not

significant. In the three generation experiment presented in Section B no significant treatment effects were present. The ratios for the G_1 progeny presented in Table XI did not follow a consistent pattern. The ratios for the 0, 5, 10, and 15 p.p.m. treatments were 1.01, 0.99, 1.05, and 0.92. The Treatment mean square for this generation (Table XII) was not significant. The ratios for the G_2 progeny presented in Table XIII were 1.00, 0.92, 0.98, and 1.00 for the 0, 5, 10, and 15 p.p.m. treatments. The Treatment mean square (Table XIV) was less than the Error mean square. However, the ratios for the G_3 generation exhibited more variation than the previous two generations. The ratios for this generation presented in Table XV for the 0, 5, 10, and 15 p.p.m. treatments are 0.99, 1.21, 1.02, and 1.05. The Treatment mean square (Table XVI) indicated the possibility of real differences being present. The results of the experiment presented in Table XXI, Section C, show that a slightly greater proportion of male offspring was produced on the 15 p.p.m. treatment. The ratios for the 0 and 15 p.p.m. treatments were 0.92 and 0.99. The Treatment mean square (Table XXII) was less than the Error mean square. The ratios for the experiment presented in Table XXVII, Section D, exhibited little variation. The ratios for the 0 and 15 p.p.m. treatments were 1.00 and 0.97. The Treatment mean square (Table XXVIII) was not significant.

The ratios of the G_2 and G_3 progeny indicate previous selection may have some influence on this ratio. The Parental Source X Treatment interaction was highly significant in the G_2 generation, whereas in the G_3 generation this interaction was not significant. The Parental Source mean square for the G_2 progeny was not significant, whereas the Parental

Source X Treatment interaction was highly significant as shown in Table XIV. The ratios of G_2 progeny presented in Table XIII show for each parental source at least one instance in which the proportion of female offspring produced exceeded at least two standard deviations from the mean of the 0 and 5 p.p.m. parental source means. The treatment on which this event occurred was not the same for the parental sources. For the parents from the 0 p.p.m. source, their ratio on the 5 p.p.m. treatment was 0.87 compared to a mean of 0.95, and for parents from the 5 p.p.m. source the greatest deviation from their source mean of 0.93 occurred on the 10 p.p.m. treatment with a ratio of 0.85. The parents from the 10 p.p.m. source exhibited the greatest variation in this ratio. On the 0 and 15 p.p.m. treatments, each produced a ratio of 0.88 and on the 5 and 10 p.p.m. treatments the ratios were 1.00 and 1.20. The mean for this parental source was 0.99. The ratios produced on these treatments by parents from the 15 p.p.m. treatment were reversed from the parents from the 10 p.p.m. source. On the 0 and 15 p.p.m. treatments the ratios of the offspring were 1.13 and 1.20, and on the 5 and 10 p.p.m. treatments the ratios were 0.92 and 0.88. The parental source mean was 1.03. The ratios for the G_3 progeny did not exhibit the variation found for the G_2 progeny. With one exception for parents originally from the 0, 5, and 10 p.p.m. sources and two exceptions for parents from the 15 p.p.m. source, the ratios presented in Table XV indicated a greater proportion of male offspring. The mean square for the Parental Source X Treatment interaction did not approach significance.

There were indications of Race differences in the experiments presented in Sections A, B, and C. In the results of the experiments

presented in Section A, the mean square for Races was highly significant. The race means were 0.88 for the (f) race and 1.06 for the (b) race. The (b) race consistently produced a greater proportion of male offspring than the (f) race in the three generation experiment presented in Section B. The ratios for the G_1 offspring produced were 0.86 and 1.10 for the (f) and (b) races. For the G_2 progeny this ratio was 0.86 and 1.09 and 0.97 and 1.18 for the G_3 progeny. In all three generations, the Race mean squares were highly significant, but the Race X Treatment mean square was not significant. These results indicate that one of the characteristics of the (b) race was to produce a greater proportion of male offspring. There were significant differences in the ratios of male to female offspring produced by the 12 races in the experiment presented in Section C. These ratios (Table XXI) ranged from 0.80 for the Oregon-H (f) race to 1.25 for the (y) race. The mean ratio for the 12 races was 0.96, whereas in the experiments of Sections A and B the differences in this ratio between the Turtex (f) and the (b) races were highly significant. In this experiment their ratios were both 0.88. The ratios produced in these experiments seem to be a particular characteristic of the race rather than being associated with resistance. The Race X Treatment interaction did not approach significance.

Thus far there is no evidence of sex differences in the resistance of the progeny to selenium. Thus, the possibility of selenium susceptibility being a sex limited character is ruled out. The observations of sex differences in susceptibility in beef cattle may be the result of management factors. The next aspect of the problem to consider is that of sex linkage. The importance of sex linkage can be determined by making

reciprocal crosses between resistant and susceptible parents and comparing the ratios of male to female offspring produced. If sex linkage is present, the heterogametic sex would be affected more severely than the homogametic sex. The evidences obtained from making reciprocal crosses between DDT resistant and susceptible strains of houseflies and fruit flies show no evidence of sex linkage.

The ratios of male to female offspring produced by the four mating systems in Experiment I (Table XXVII, Section D) were all within one standard deviation of one another. The ratios produced by the four mating systems are as follows: R X R 1.01, S X S 0.96, R X S 0.96, and S X R 0.98. The mean squares for Mating Systems were not significant (Table XXVIII). These results indicate that susceptibility to selenium poisoning is not affected by sex linkage.

However, the results of Experiment II presented in Table XXIX, Section D, in which the effect of previous selenium history of the parents was studied on these mating systems, exhibit more mating system variation than in Experiment I. In this experiment, those matings in which the female was the susceptible parent tended to produce a greater proportion of male offspring. The ratios for the mating systems were as follows: 0.92 for the R X R, 0.94 for the S X S, 1.04 for the R X S, and 0.90 for the S X R matings. The mean square for Mating Systems (Table XXX) was not significant. The variation in the ratios produced by parents in the different source combinations did not exhibit much variation. The ratios for the 0 X 0 was 0.94, for the 15 X 15 0.95, for the 0 X 15 0.91 and 1.01 for the 15 X 0 combination. The mean square for Parental Source combination was not significant. The interaction mean square for these two sources of variation did not approach significance.

The results of studying the effect of the treatments on the ratio of male to female offspring indicate no difference and no evidence of sex linkage. Thus, it can be concluded that genes for reproductive performance in both the non-selenized and selenized environments are located on the autosomes rather than on the sex chromosomes.

The observations reported by Dinkel et al. (10) that bulls exhibit more severe symptoms of selenium poisoning made it desirable to study the influence of the sex of the resistant or susceptible parent on the resistance exhibited by the progeny. This was studied by comparing the performance of four mating systems, R X R, S X S, R X S, and S X R to determine what influence, if any, the sex of the resistant parent may have on the resistance of their progeny. The results of the experiment presented in Table XXIII, Section D, in which the performance of four mating systems on two treatments were compared indicate that each parent contributed an equal amount of resistance to the progeny. There were also indications from this experiment that hybrid vigor may also contribute to the resistance of offspring. The significant ($P \leq 0.05$) Mating System mean square (Table XXIV, Section D) is reflected in the means for the four mating systems. The two reciprocal matings, R X S and S X R, produced a mean of 112.0 and 95.1 offspring compared to the performance of the R X R and S X S matings which produced a mean of 94.4 and 75.2 offspring per female parent. The performance of the reciprocal matings on the 0 p.p.m. treatment exhibited a greater degree of hybrid vigor than these matings on the 15 p.p.m. treatment. The reciprocal matings on the 0 p.p.m. treatment produced 167.8 and 135.4 offspring per female parent compared to 129.5 for the R X R and 110.5 offspring per female parent for the S X S mating.

On the 15 p.p.m. treatment, the reciprocal matings produced 56.3 and 56.1 offspring per female parent compared to 59.2 and 39.9 offspring per female parent for the R X R and S X S matings. The results of these matings indicate that the progeny produced from matings in which at least one parent is resistant may exhibit superior resistance to those in which both parents are susceptible.

The results of this experiment are similar to those observed in studies of insecticide resistance in houseflies and fruit flies. The results of reciprocal crosses between DDT resistant and susceptible strains of houseflies made by Harrison (18) show the F_1 progeny were intermediate in their resistance to the parental strains and the resistance of the F_2 was quite variable. There was no indication from these studies of a difference in tolerance transmission, whether emanating from matings between two resistant parents or between a resistant and susceptible strain. Similarly, Crow (6) and Pimental et al. (33) found the F_1 progeny produced from reciprocal crosses between DDT resistant and susceptible strains of houseflies were intermediate to the resistance exhibited by their parents. Busvine and Khan (4) found similar results in their studies of BHC resistance in houseflies. The absence of sex differences in resistance to either insecticides or selenium in insects may also be valid for domestic animals assuming a similar hereditary mechanism.

While the results of the experiment presented in Table XXIII, Section D, indicate that each parent contributes an equal amount of resistance to the progeny, the results of the experiment presented in Table XXV, Section D, indicate that the female parent is more sensitive

to the presence of selenium reproductive-wise than the male. It will be recalled from Section D that the non-selenized parents were obtained from the laboratory stocks and the selenized parents were obtained from stocks which had been maintained on selenium for several generations. The means for the Parental Source combinations presented in Table XXV show that the matings in which the females were from the non-selenized stocks produced more offspring than those from the selenized stocks. The means for the 0 X 0 and 15 X 0 combinations were 43.1 and 45.3 compared to 29.0 and 36.6 offspring per female for the 15 X 15 and 0 X 15 combinations. The mean square for Parental Sources presented in Table XXVI indicate these differences may be real. The means for the 0 X 0, 15 X 0, and 0 X 15 combinations compared with the 15 X 15 combination indicate that matings in which at least one parent is non-selenized produce more offspring than matings in which both parents are from selenized stocks.

The mating system means indicate that the level of resistance of the female parent had considerable influence on the performance of the four mating systems. The matings in which the female was the resistant parent, R X R and S X R, produced 44.4 and 48.2 offspring per female parent compared to 27.2 and 30.1 offspring per female parent for the S X S and R X S matings in which she was the susceptible parent. The significant Mating Systems mean square reflects the variation in performance contributed by differences in the level of resistance of the female.

The absence of a significant Parental Source X Mating System interaction indicates the level of resistance and previous exposure to selenium of the female parent plays an important role. The performance of the

four parental source combinations exhibited, with one exception, a similar pattern for the R X R and S X R matings. For the R X R mating, the O X O and 15 X O combinations produced a mean of 52.7 and 53.8 offspring per female parent. This performance is contrasted with that of the 15 X 15 and O X 15 combinations producing 39.9 and 31.2 offspring per female parent. A comparison of the performance of these matings shows that previous selenium history may affect the female to a greater extent than the male parent. The S X R mating also exhibited a somewhat similar pattern. The O X O and 15 X O combinations produced 43.9 and 52.8 offspring per female parent and the 15 X 15 combination produced 27.0 offspring per female parent. The O X 15 combination however, produced a mean of 64.8 offspring per female parent. The performance of this particular combination was superior to all of the parental source-mating system combinations making up this experiment.

The performance of the S X S and R X S matings compared to the performance of the R X R and S X R indicates the effect that selenium may have on the reproductive performance of the susceptible female parent. The mean performance of these combinations for the S X S matings were 19.8 for the O X O, 32.6 for the 15 X 15, 16.6 for the O X 15 and 41.1 offspring per female parent for the 15 X O combinations. The variation in performance on the O X O and 15 X 15 combinations indicates some selection for resistance may have occurred in the latter combination. Since the offspring in this experiment were produced on 15 p.p.m. selenium, the performance of O X O combination may indicate the performance of the unselected susceptible strain, whereas their performance on the 15 X 15 combination indicates the effect of their previous selection for

resistance. The variation in performance of the 0 X 15 and 15 X 0 combinations reflect the effect of 15 p.p.m. selenium on the performance of the susceptible selenized female parent.

The performance of the different parental source combinations for the R X S mating also exhibited a similar pattern. The means for the parental source combinations were 45.6 for the 0 X 0, 17.7 for the 15 X 15, 26.4 for the 0 X 15, and 30.9 offspring per female parent for the 15 X 0 combination. The variation between the 0 X 0 and 15 X 15 combinations indicates the effect of 15 p.p.m. selenium on the reproductive performance of the susceptible selenized female parent. While the differences were not great between the 0 X 15 and 15 X 0 combinations, the superior performance of the latter combination indicates that more offspring were produced by the combination in which the female parent was the non-selenized parent.

Thus, recognizing individual variation which may have been due to chance, the results from this experiment indicate that the reproductive performance of the female is more sensitive to selenium than that of the male. Furthermore, there were indications that the females from the susceptible strain were affected to a greater extent by selenium than females from the resistant strain. These results did not indicate precisely at what stage this effect occurred. These results may have been the result of a selenized female producing either fewer eggs or a greater proportion of infertile eggs than the non-selenized female. In addition, the possibility exists that larvae produced from eggs laid by the selenized female were weaker with consequently higher mortality before emergence.

The performance of the $R_0 \times R_{15}$ and the $S_0 \times R_{15}$ combinations are of interest since these are the combinations which may be expected to occur the most frequently in the affected areas, assuming the purchased sires are either resistant or susceptible and are mated to resistant females. While the performance of the $R_0 \times R_{15}$ combination was near that of the experimental mean, the $S_0 \times R_{15}$ combination produced nearly twice as many offspring per female as the $R_0 \times R_{15}$ combination. The superior performance of the $S_0 \times R_{15}$ may indicate the presence of specific combining ability, since by definition specific combining ability exists when the performance of a specific cross is superior to the average of all possible matings in a particular population.

The performance of the four mating systems in which both parents were from the non-selenized source ($O \times O$) were similar to those for Experiment I presented in Table XXIII, Section D. The performance of the reciprocal crosses indicated the presence of hybrid vigor. The means for the reciprocal crosses were 45.6 and 43.2 offspring per female parent and the $R \times R$ and $S \times S$ matings produced 52.7 and 19.8 offspring per female parent. The small variation between the reciprocal crosses and their approaching the performance of the $R \times R$ mating is exactly the same pattern as found in Experiment I.

The discrepancy between the results of Experiment I and the portion of Experiment II in which both parents were obtained from non-selected stocks and those of Experiment II in which the parents were obtained from different treatment sources may be the result of the effect of prior selenium exposure impairing reproductive potential of the female. Evidence of a similar nature has been reported in insecticide studies.

Norton (32) and Pimental et al. (33), working independently, concluded from their respective studies of DDT resistance in the housefly that the female parent influenced the resistance of the progeny to a greater extent than the male, but neither concluded this was evidence of sex linkage. The conclusion arrived at by Crow (6) from studies of DDT resistance in D. melanogaster that the two major autosomes contribute an equal amount of resistance, but that sex linked and cytoplasmic contributions are not important, may be valid for the situation in which both parents are from the same treatment source.

The evidence obtained from experiments in which the performance of parents was subjected to one or more generations of exposure to selenium indicates that selenium may have affected the reproductive potential of these parents. Similar evidence has been obtained from studies of the effect of selenium on reproduction in rats and poultry. The observations of Franke and Tully (14, 16) and Franke et al. (15) on the manner in which selenium effects hatching percentage in poultry may also be applicable to the results found in this study. The findings of Westfall (43) studying the placental transmission of selenium and Rosenfeld and Beath (36) studying the effect of different levels of selenium on reproduction in the rat may in part help interpret these results. There was evidence of placental transmission of selenium in the female rat and that levels of 7.5 p.p.m. selenium restricted reproduction in the female but did not affect the fertility of the male.

The discussion thus far has been concerned with specific aspects of the general problem of selenium toxicity. The rancher in the affected area, however, will rarely have an opportunity to consider each of these

aspects separately when making his choice of herd replacements. Since the individual is the smallest unit he can select, his selection is based primarily upon the phenotypic expression of resistance or susceptibility. Since the phenotype on which he selects is the sum of a large number of genetic and environmental factors, the success of his selection is going to be dependent upon how accurately the phenotype reflects the genotype. Hence, the choice of his breeding program is going to be dependent to a large extent upon the heritability of this trait, since heritability provides a measure of this reflection. To provide recommendations of a breeding program for increasing resistance, estimates of heritability of reproductive fitness and resistance were calculated in Section E. These estimates are discussed in terms of the results of the experiments presented in Sections A, B, C, and D.

The estimates of heritability of reproductive fitness calculated in Section E indicated that a considerable portion of the differences between individuals was hereditary in origin. The estimates in the broad sense did not vary greatly from the estimates in the narrow sense. There were indications, however, that treatment differences were present in the estimates in the broad sense. The estimates of heritability of resistance in the broad sense were quite low indicating hereditary differences between individuals contributed only a small portion to the total variance.

Heritability in the narrow sense by definition includes only the genic variation between individuals. However, in practice this estimate is often between the broad and narrow sense since dominance, epistatic and environmental effects may be included in the genic variance. The

method of calculating heritability is dependent upon how closely the phenotypic resemblance parallels the genetic resemblance. These are often, for example as in this study, calculated from the Analysis of Variance. Since only in a few special cases can these resemblances be known, genetic resemblance is inferred from the relationship. In random mating populations, the relationship between full sibs is 0.50 and the relationship between half sibs is 0.25. Equating these relationships to unity provides the factors used to multiply the resemblances for heritability. In the case of full sibs, the factor is two and for half sibs the factor is four.

The estimates of the heritability of reproductive fitness in the narrow sense were all greater than 0.20 indicating that mass selection for this trait in a selenized environment may be the most effective means of breeding for resistance. The estimates calculated from the components of variance are presented in Table XXXII, Section E. A comparison of these components indicate that sire differences contributed more variation than dam differences. The mean square for Sires, while not significant, indicated real differences may be present. Of the half sib estimates, the one based on the maternal half sib correlation of 0.38 may be the most reliable since the fiducial limits of 0 and 310.01 for the dam component were smaller than the limits of 0 and 357.95 for the sire component. The estimate based on the paternal half sib correlation was 0.50. The estimate of 0.43 based on the full sib correlation while intermediate to the estimates based on the half sib correlations includes some of the dominance and epistatic deviations.

A large component of the trait identified as reproductive fitness herein may include egg production. Consequently, a large proportion of the variation found herein may have been the result of differences in egg production between individuals. The estimates of reproductive fitness calculated were in relatively close agreement with those obtained in studies of the heritability of egg production in poultry. Lerner (23), summarizing the results of several studies of this trait in poultry, reports estimates ranging from 0.29 to 0.35.

The second concept of heritability is that of heritability in the broad sense. The function of the genotype as a unit within the individual is considered heritability in the broad sense. It is used in this manner when contrasting the hereditary variation with the environmental. However, the genotype is not transmitted as unit as the result of segregation and recombination. Combinations which may produce certain effects in the parental generation may only be transmitted in part, if at all. Thus, the essential difference between heritability in the broad and narrow sense is that the latter estimate includes only the genic variation (σ^2), whereas the former estimate includes the hereditary variance from all sources. The second method of calculating heritability was for the purpose of providing an indication of the validity of the estimates in the narrow sense.

The estimates of reproductive fitness in the broad sense on both the 0 and 15 p.p.m. treatments indicate a relatively large proportion of the total variance was contributed by differences in heredity between individuals. The estimates of reproductive fitness in the broad sense

were generally in the same range as the estimates of reproductive fitness in the narrow sense. The differences between the estimates of reproductive fitness in the narrow sense and the broad sense may include uncontrolled environmental variations since the experiments from which the two types of estimates were calculated were not contemporary.

The Analyses of Variance from which the hereditary components (Races) of reproductive fitness and the environmental components (Error) are presented in Tables XXXIII and XXXIV, Section E. The data from which these estimates were calculated were obtained from the two generation experiment presented in Section C. The estimates were calculated on an intra-generation basis in order to prevent the introduction of bias resulting from generation differences. Estimates based on the combined results of each generation were also calculated to study the influence of generation differences. The pattern of the estimates indicated the reproductive performance in a non-selenized environment is dependent to a greater extent upon environmental differences, whereas in the selenized environment hereditary differences between individuals appear to be more important.

The ratio between the hereditary and environmental components in the first generation for the 0 and 15 p.p.m. treatments presented in Tables XXXIII and XXXIV, Section E, were essentially the same. This indicates that in the first generation, irrespective of treatment differences, hereditary differences between individuals were equally as important. In each case, the value of the hereditary component varied little from the environmental component. The estimate of 0.49 ± 0.70 for each treatment indicates that hereditary differences between individuals

contributed approximately half of the total variance. It will be recalled from Section C that the treatment differences in the first generation were not significant, apparently as the result of unfavorable temperatures. The probability then exists that the unfavorable environmental conditions exerted a more severe test of the genotypes making up this particular experiment than did 15 p.p.m. selenium. This concentration of selenium was found to be highly toxic in Sections A, B, C, and D.

In the second generation, the ratios for each treatment exhibited a widely divergent pattern. Tables XXXIII and XXXIV, Section E, show that environmental differences contributed a greater proportion of the total variance on the 0 p.p.m. treatment, whereas on the 15 p.p.m. treatment the opposite occurred. The considerable increase in the environmental contribution for the 0 p.p.m. treatment in this generation indicates the marked increase in reproductive performance was due to this factor and not to genetic influences. The estimate of 0.20 ± 0.47 for this treatment indicates that only 20 percent of the total variance was contributed by hereditary differences compared to nearly 50 percent for the first generation. The ratios for the 15 p.p.m. treatment in this generation indicated that selenium exerted a much more severe test on the genotypes despite the more favorable temperature conditions. The estimate of 0.70 ± 0.84 indicates that 70 percent of the total variance was contributed by hereditary differences compared to approximately 50 percent in the first generation.

A comparison of the hereditary and environmental components for two generations combined (Table XXXV, Section E) show that environmental differences exerted a much greater effect on the performance of parents

on the 0 p.p.m. treatment than for those on the 15 p.p.m. treatment. The estimate for the two generations combined on the 0 p.p.m. treatment was 0.06 ± 0.24 which indicates that for the two generations only six percent of the total variance was due to hereditary differences. On the other hand, the estimate of 0.67 ± 0.82 for the 15 p.p.m. treatment indicates that hereditary differences contributed 67 percent of the total variance in the two generations.

These estimates despite their wide standard errors indicate that differences in heredity between individuals assumes considerable importance particularly in an unfavorable environment. A comparison of these estimates with those obtained in the narrow sense indicate that a rather substantial portion of the variance may be due to additive acting genes. These estimates, however, demonstrate what may occur when wide environmental variation is present. The proportion of the hereditary variance to the total variance was essentially the same for both treatments in the first generation. In the second generation, however, this relationship changed apparently as the result of temperature differences. The ratios between the hereditary and environmental components for the two generations combined on each treatment further demonstrate the effect of environment on the ratio of the hereditary variance to the total variance.

The estimates of heritability of resistance in the broad sense exhibited considerable between generation differences. These estimates were calculated on a within generation basis as well as for two generations combined. The Analyses of Variance and components of variance presented in Table XXXVII, Section E, were calculated from the data presented in Table XIX, Section C. A comparison of the components of

variance for each generation indicates that the hereditary contribution for the first and second generations was essentially the same. However, the environmental component in the second generation was approximately six times that of the first generation. As a result, the first generation estimate of 0.25 ± 0.50 was approximately six times that of the second generation estimate of 0.04 ± 0.24 . When the results of the two generations were combined, an estimate of 0.05 ± 0.24 was obtained. The components of variance for the latter estimate was obtained from the Analysis of Variance presented in Table XX, Section C. The generation differences apparently reflect environmental variation rather than changes in gene frequency resulting from one generation selection.

The wide standard errors associated with these estimates limit their validity. Two of the three estimates of resistance calculated were in the same general area as reported for disease resistance in poultry. Lush et al. (26), in a study involving more than 20,000 leghorn hens using directly observed percentages, obtained estimates of 0.083 for resistance to total mortality, 0.053 for resistance to the leucosis complex, and 0.034 for resistance to death from other causes than leucosis. Assuming a similar mechanism may be involved for selenium resistance as in disease resistance, selection for selenium resistance alone may be a relatively slow process. The indication that environment may play an important role in the expression of selenium resistance may be of considerable significance to the breeder.

The estimates of heritability of reproductive fitness and resistance presented in Section E indicate that at least a part of the variation found in Sections A, B, C, and D is due to difference in heredity between

individuals. Since at least part of the variation found herein was due to hereditary differences, breeding for selenium resistance may be feasible in areas where the use of preventative agents are precluded by management factors. Once the breeder has decided that breeding for resistance is worth the effort, his next step is to utilize such methods which will yield the maximum progress with minimum expense. The progress he achieves will be dependent upon the amount of genic variance, i.e., heritability in the narrow sense that is available. There are several factors which may cause heritability to be low. Among those listed by Lush (25) are low genic variance, the effects of dominance and epistasis, and genetic and environmental interactions. The degree to which each is present will to a large extent determine the choice of the breeding system and selection program.

The differences in the estimates of reproductive fitness in the narrow and broad sense on the 15 p.p.m. treatment indicate that hereditary differences between individuals are more important for reproductive fitness than for selenium resistance. Thus, one of the reasons why the heritabilities of resistance were low may be due to the fact that little genic variance is present. One reason why genic variance may be low is that genes which confer resistance may exist in a neutral state or exhibit a selective disadvantage in the absence of selenium. This has been postulated in studies of insecticide resistance. Since the individual must reproduce before resistance or susceptibility can be expressed, genotypes conferring superior reproductive fitness may also have an additional function of expressing resistance in the appropriate environment. Thus, pleiotropy may be indicated.

Attempts at correlating certain physical attributes with insecticide resistance have not proven entirely successful, whereas studies of disease resistance in poultry have indicated that resistance to the leucosis complex and constitution may be correlated. Lush et al. (26) reported a correlation of $r = 0.54$ between resistance to the leucosis complex and resistance to death from other causes. Similarly, Hutt and Cole (19) selecting for resistance to the leucosis complex found that egg production and viability improved concurrently. In this study a phenotypic correlation of $r = 0.32$ was found between reproductive fitness and resistance in the experiment presented in Section C. Indirect evidence was observed in the experiments presented in Section D that parental vitality and reproductive performance on the 15 p.p.m. treatment may be related. It will be recalled from Experiment I that the Experiment X Treatment interaction mean square was highly significant ($P \leq 0.01$). Table XXII shows that, for both Experiments A and B, the performance of the four mating systems on the 0 p.p.m. treatment was not affected by parental stock differences. However, on the 15 p.p.m. treatment, the toxicity exhibited by this treatment in Experiment A was much more severe than in Experiment B. This variation may have been the result of differences in parental vitality since the parents for Experiment A were obtained from nearly exhausted laboratory stocks.

The rancher's choice of a breeding program will be dependent upon the criterion he uses to measure resistance. If the trait is highly hereditary, individual selection may be the most effective means of selecting for resistance. On the other hand, the low heritability of resistance found herein does not mean that only a small gain in progress

is possible. It could mean that the hereditary variability is due to a large number of minor factors and that the favorable allele is generally the uncommon one. Thus, potential progress may exceed the current limit. Under these conditions direct observation will give only a small clue as to the individual's transmitting ability. Preliminary selection based on attention to collateral relatives followed by selection based on a progeny test may be the most effective under these conditions.

Dominance and epistasis may have contributed to the presence of hybrid vigor in Experiment I, Section D. If dominance and epistasis contribute a large proportion to the total variance, a reduction in heritability would result. As in the case of low genic variance, direct observation will give little indication as to the individual's transmitting ability. Where dominance is important, preliminary selection should be based upon the performance of collateral relatives followed by selection based on a progeny test. Ultimately, as the program continues, a greater proportion of the total variance will become dependent upon the rare recessives present. At this point an inbreeding program will become necessary in order to uncover the rare recessives. Inbreeding accompanied by selection will also be important where epistatic effects are prominent. However, it will become an increasingly important tool since the ultimate goal is to develop lines which are homozygous for some special combination. The amount of additive variance present in addition to the epistatic effect will determine the form of inbreeding. If a large proportion of the variance is additive, linebreeding may be the most important. On the other hand, if little of the variance is additive, those forms of inbreeding which create as many partially inbred lines as possible may be indicated.

An occasional outcross may be necessary to maintain the desired level of production. In addition, where overdominance is important crossing of the lines to tester stocks may be necessary.

The final factor which may cause heritability to be low is the interaction between heredity and environment. The variance contributed by this interaction may assume considerable importance in a study of this nature. The only way to determine the individual's resistance or susceptibility is by testing it in a selenized environment. The importance of this interaction can be ascertained by testing representatives of the same race or sire group in two environments, non-selenized and selenized. A test of the significance of the Group X Treatment interaction mean square will indicate whether genotypes favored in one environment differ from those tested in the second environment.

The results of the experiments presented in Sections A, B, and C indicate that interactions between heredity and environment are present for reproductive fitness. With the exception of the G_2 and G_3 generations in Section B, the Race X Treatment interaction mean squares were significant. The differences in genotype are represented by the races used in the experiments and environmental differences by the selenium treatments. The significant interactions resulted from a differential level of reproductive performance of the races on the various selenium treatments. In addition to the interaction between races and treatment, the Race X Generation interaction was also significant in Section C. This indicates that temperature differences between the first and second generation contributed to the differential reproductive performance of the 12 races between the two generations.

The presence of the significant interactions between races and treatments and races and generations may explain the wide variation in the estimates of the heritability of reproductive fitness and resistance in the broad sense. Since the estimates in the broad sense are essentially contrasting the hereditary with the environmental, the ratio between these two was affected by treatment differences within the same experiment. However, the laboratory temperatures which varied between the first and second generation also affected this ratio. Consequently, the ratio of the hereditary variance to the total variance for each treatment in the first and second generations exhibited a widely divergent pattern. This effect was also present in the estimates of the heritability of resistance.

This type of interaction may assume considerable importance in breeding for selenium resistance since the presence of selenium may be necessary for the expression of genotypes conferring resistance. Under these conditions, the optimum method of breeding for resistance will be the testing of a large number of genotypes in the environment in which they are to be kept and preserving those which manifest the desired trait to a superior degree. Following this, weighing emphasis on individual selection by pedigree or progeny testing while working on the broadest genetic base may be necessary. When progress slackens, further advance may be possible by development and crossing of inbred lines.

SUMMARY

The purpose of the experiments presented herein was to study the inheritance of selenium resistance in the fruit fly, Drosophila melanogaster. These studies were instituted to complement a study of a similar nature being conducted with beef cattle in a known seleniferous area in South Dakota. The criterion used to measure selenium toxicity was the mean number of offspring produced per female parent. The experiments presented herein were designed primarily to study, (1) the toxicity of various concentrations of selenium, (2) the effect of different selection intensities on subsequent performance, (3) race differences and resistance, (4) the influence of sex on resistance, and (5) to obtain estimates of the heritability of reproductive fitness on non-selenized and selenized treatments and estimates of the heritability of resistance.

The toxicity of four selenium treatments, 0, 5, 10, and 15 p.p.m., was not linear. The 5 and 10 p.p.m. treatments did not result in any appreciable degree of toxicity. The 15 p.p.m. treatment was found to be highly toxic in nearly every experiment. It was concluded that a threshold in toxicity was present between the 10 and 15 p.p.m. treatments.

The results of a three generation experiment conducted for the purpose of studying the subsequent performance of parents subjected to varying intensities of selection indicate that reproductive performance in a selenized environment may be in part due to differences in heredity. Preliminary experiments indicated that the parental population is comprised of a mixture of relatively resistant and susceptible individuals. There was no conclusive evidence as to whether selection favored reproductive

fitness or resistance from these experiments since in one generation, it appeared that selection had favored both, whereas the results of the subsequent generation indicated that only reproductive fitness had been favored.

There were highly significant race differences present for reproductive performance on selenium and real differences may be present in their resistance to selenium. The results of testing 12 races, five wild and seven mutant, indicated that differences in reproductive performance and resistance were the result of individual differences rather than of the morphological characters which identify the various races tested. A low positive correlation was found between reproductive fitness and resistance.

The results of the studies concerned with the influence of sex indicated that both sexes are equally resistant and that they contribute an equal amount of resistance to the progeny. The results of analyzing the ratio of male to female offspring produced in the experiments making up this study revealed no significant treatment differences, however, there were indications that race differences were present. A comparison of the performance of reciprocal matings between resistant and susceptible stocks indicated that both parents contribute equally to the resistance of their progeny. The performance of these reciprocal matings indicated that hybrid vigor is present for resistance. Evidence obtained from a series of matings in which all combinations of resistant and susceptible female and male parents from non-selenized and selenized stocks indicated that selenium impaired the reproductive performance of the female to a greater extent than that of the male parent. The females from the

resistant stocks were affected to a lesser degree than those from susceptible stocks.

The estimates of the heritability of reproductive fitness in the narrow and broad sense were higher than the estimates of resistance in the broad sense. The estimates of reproductive fitness in the narrow sense based upon maternal and paternal half-sib correlations and full-sib correlations were 0.38, 0.50, and 0.43, respectively. The estimates of the heritability of reproductive fitness in the broad sense were as follows: first generation, 0.49 ± 0.70 for the 0 and 15 p.p.m. treatments; second generation, 0.20 ± 0.47 for the 0 p.p.m. treatment and 0.70 ± 0.84 for the 15 p.p.m. treatment. The estimate for the two generations combined was 0.06 ± 0.24 for the 0 p.p.m. treatment and 0.67 ± 0.82 for the 15 p.p.m. treatment. The estimates of the heritability of resistance were as follows: 0.25 ± 0.50 for the first generation, 0.04 ± 0.24 for the second generation and 0.05 ± 0.24 for the two generations combined.

LITERATURE CITED

- (1) Anderson, E. H., "Growth Requirements of Virus Resistant Mutants of Escherichia coli, Strain "B", Proceedings of the National Academy of Science, vol. 32, pp. 120-128, University of Chicago Press, Chicago, Ill., 1946.
- (2) Bridges, Calvin, and Katherine Brehme, "The Mutants of Drosophila melanogaster," Carnegie Institute Publication No. 552, Carnegie Institute, Washington, D. C., 1944.
- (3) Bross, Irwin, "Fiducial Intervals for Variance Components," Biometrics, vol. 6, pp. 136-144, American Statistical Society, Washington, D. C., 1950.
- (4) Buavine, J. R., and Khan, J. H., "Inheritance of BHC Resistance in the Housefly," Transactions of the Royal Society of Tropical Medicine and Hygiene, vol. 49, pp. 455-459, Mansion House, London, England, 1955.
- (5) Cochran, William, and Cox, Gertrude, Experimental Designs, pp. 126-127, John Wiley and Sons, New York, New York, 1950.
- (6) Crow, James F., "Some Genetic Aspects of Selection for Resistance," National Research Council Publication No. 219, pp. 72-78, National Academy of Science, Washington, D. C., 1954.
- (7) _____ "Analysis of a DDT Resistant Strain of Drosophila," Journal of Economic Entomology, vol. 47, pp. 393-398, Entomological Society of America, Washington, D. C., 1954.
- (8) Demerec, M., "Production of Staphylococcus Strains Resistant to Various Concentrations of Penicillin," Proceedings of the National Academy of Sciences, vol. 31, pp. 16-24, University of Chicago Press, Chicago, Ill., 1945.
- (9) _____ "Development of Bacterial Resistance to Chemicals," National Research Council Publication No. 219, pp. 67-69, National Academy of Sciences, Washington, D. C., 1952
- (10) Dinkel, C. A., Minyard, J. A., Whitehead, E. E., and Olson, O. E., "Agricultural Research at the Reed Ranch Field Station," Experiment Station Progress Report, Circular No. 135, South Dakota State College, Brookings, S. Dak., 1957.
- (11) Dobzhansky, Th., Genetics and Origin of the Species, 3rd Ed., (rev), pp. 95-96, Columbia University Press, New York, New York, 1951.

- (12) Franke, Kurt W., "A New Toxicant Occurring Naturally in Certain Samples of Plant Food Stuff. I. Results Obtained in Preliminary Feeding Trials," Journal of Nutrition, vol. 8, pp. 597-608, American Institute of Nutrition, Wistar Institute of Anatomy and Biology, Philadelphia, Penn., 1934a.
- (13) _____, and Tully, W. C., "A New Toxicant Occurring Naturally in Certain Samples of Plant Food Stuff. V. Low Hatchability Due to Deformities in Chicks," Poultry Science, vol. 15, pp. 316-318, Poultry Science Association, Texas A and M College, College Station, Texas, 1935.
- (14) _____, and Potter, Van R., "A New Toxicant Occurring Naturally in Certain Samples of Plant Food Stuff. IX. Toxic Effects of Orally Injected Selenium," Journal of Nutrition, vol. 10, pp. 213-221, American Institute of Nutrition, Wistar Institute of Anatomy and Biology, Philadelphia, Penn., 1935.
- (15) _____, and Tully, W. C., "A New Toxicant Occurring Naturally in Certain Samples of Plant Food Stuff. VII. Low Hatchability Due to Deformities in Chicks Produced From Eggs Obtained from Chicken of Known History," Poultry Science, vol. 16, pp. 273-279, Poultry Science Association, Texas A and M College, College Station, Texas, 1936.
- (16) _____, Moxon, A. L., Poley, W. E., and Tully, W. C., "Monstrosities Produced by the Injection of Selenium into Hens Eggs," The Anatomical Record, vol. 65, pp. 15-22, Wistar Institute of Anatomy and Biology, Philadelphia, Penn., 1936.
- (17) _____, Painter, N., and Page, E., "A Study of Toxicity and Selenium Content of Seleniferous Diets with Statistical Considerations," Cereal Chemistry, vol. 15, pp. 1-24, American Association of Cereal Chemists, University Farm, St. Paul, Minn., 1937.
- (18) Harrison, Mary C., "Inheritance of Resistance in the Housefly, Musca Domestica L.," Nature, vol. 167, pp. 855-856, American Naturalist Association, Washington, D. C., 1951.
- (19) Hutt, F. B., and Cole, R. K., "Control of Leucosis in the Fowl," Journal, American Veterinary Medical Association, vol. 131, pp. 491-495, American Veterinary Medical Association, Chicago, Ill., 1957.
- (20) Johnson, E. F., Bogart, Ralph, and Lindquist, A. W., "The Resistance to DDT by Houseflies. Some Genetic and Environmental Factors," Journal of Heredity, vol. 45, pp. 177-182, American Genetics Association, Baltimore, Md., 1954.

- (21) King, James C., "The Genetics of Resistance to DDT in Drosophila melanogaster," Journal Economic Entomology, vol. 47, pp. 387-393, Entomological Society of America, Washington, D. C., 1954.
- (22) _____, "Evidence of the Integration of the Gene Pool from Studies of DDT Resistance in Drosophila," Cold Spring Harbor Symposium on Quantitative Biology, vol. 20, pp. 311-317, Unknown, 1955.
- (23) Lerner, I. M., and Cruden, D. M., "The Heritability of Accumulative Annual Egg Production," Poultry Science, vol. 27, pp. 67-68, Poultry Science Association, Texas A and M College, College Station, Texas, 1948.
- (24) _____, Population Genetics and Animal Improvement, University Press, Cambridge, England, 1950.
- (25) Lush, Jay L., The Genetics of Populations, Mimeographed Textbook, Iowa State College, Ames, Iowa, 1948.
- (26) _____, Lamoreux, W. F., and Hazel, L. N., "The Heritability of Resistance to Death in the Fowl," Poultry Science, vol. 27, pp. 375-388, Poultry Science Association, Texas A and M College, College Station, Texas, 1948.
- (27) Luria, S. E., and Delbruck, M., "Mutations of Bacteria from Virus Sensitivity to Virus Resistance," Genetics, vol. 28, pp. 491-511, Genetics, Baltimore, Md., 1943.
- (28) Merrel, D. J., and Underhill, J. C., "Selection for DDT Resistance in Inbred and Laboratory Strains of Drosophila melanogaster," Journal Economic Entomology, vol. 49, pp. 300-306, American Entomological Association, Washington, D. C., 1956.
- (29) Metcalf, Robert L., "Physiological Basis for Insect Resistance to Insecticides," Physiological Reviews, vol. 35, pp. 197-232, American Physiological Society, Washington, D. C., 1955.
- (30) Moxon, A. L., "Alkali Disease or Selenium Poisoning," Agricultural Experiment Station Bulletin No. 311, South Dakota State College, Brookings, S. Dak., 1937.
- (31) Munsell, Hazel, Devaney, Grace M., and Kennedy, Mary E., "Toxicity of Food Containing Selenium as Shown by Its Effect on the Rat," Technical Bulletin No. 534, United States Department of Agriculture, Washington, D. C., 1936.
- (32) Norton, Robert J., "Inheritance of DDT Tolerance in the Housefly," Contribution, Boyce Thompson Institute, vol. 17, pp. 105-126, Unknown, 1953.

- (33) Pimental, David, Schwardt, H. H., and Dewey, J. E., "The Inheritance of DDT Resistance in the Housefly," Annals, Entomological Society of America, vol. 47, pp. 208-213, Unknown, 19__.
- (34) Poley, W. E., Wilson, W. O., and Moxon, A. L., "The Effects of Selenized Grain on the Growth of Chicks," Poultry Science, vol. 20, pp. 171-179, Poultry Science Association, Texas A and M College, College Station, Texas, 1941.
- (35) Pratt, John J., and Babers, Frank H., "The Resistance of Insects to Insecticides. Some Differences Between Strains of Houseflies," Journal Economic Entomology, vol. 46, pp. 864-869, Entomological Society of America, Washington, D. C., 1953.
- (36) Rosenfeld, I., and Beath, O. A., "The Effects of Selenium on Reproduction in Rats," Proceedings, Experimental Biology and Medicine, vol. 87, pp. 295-297, Unknown, 1954.
- (37) Snedecor, George W., Statistical Methods, 5th Ed., p. 382, Iowa State College Press, Ames, Iowa, 1956.
- (38) Shoffner, R. N., "The Heritability of Egg Production," Poultry Science, vol. 25, p. 412, Poultry Science Association, Texas A and M College, College Station, Texas, 1946.
- (39) _____, and Sloan, H. J., "Heritability Studies in the Domestic Fowl," Official Report, Eighth World's Poultry Congress, vol. 1, pp. 269-281, Copenhagen, Denmark, 1948.
- (40) Wahlstrom, R. C., Kestera, L. D., and Olson, O. E., "The Effects of Arsanilic Acid and 3-Nitro-4-Hydroxyphenylarsonic Acid on Selenium Poisoning of the Pig," Journal of Animal Science, vol. 14, pp. 105-110, American Society of Animal Production, 1955.
- (41) Sokal, Robert, and Hunter, Preston, "Reciprocal Selection for Correlated Quantitative Characters," Science, vol. 119, pp. 649-651, American Association for the Advancement of Science, Washington, D. C., 1954.
- (42) _____ "A Morphometric Analysis of DDT Resistant and Non-Resistant Houseflies," Annal, Entomological Society of America, vol. 48, pp. 499-507, Unknown, 1955.